Session text above this point is available in the transcript, available from the **Transcript Assistant** on the toolbar.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001085207	A2	20011115	WO 2001-US14796	20010507
WO 2001085207	A3	20020711		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 20020004041	A1	20020110	US 2001-804584		20010312
AU 2001059624	A	20011120	AU 2001-59624		20010507
PRIORITY APPLN. INFO.:			US 2000-565958	A	20000505
			US 2001-804584	A2	20010312
			US 1999-251896	A2	19990219
			WO 2001-US14796	W	20010507

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose methodol. for modulating the cellular immune response to a pre-selected antigen, either ex vivo or in vivo, whereby dendritic cell maturation is permitted to occur in the absence (or presence) of effective CD4+ T-cell help. The authors also disclose that phagocytosis by dendritic cells was mediated via β5-integrin. In one example, the authors demonstrate that an anti-influenza cytotoxic T-cell response was enhanced on incubation of syngeneic T-cells with dendritic cells and apoptotic monocytes infected with influenza A virus. In a second related example, anti-influenza cytotoxic T-cell response was suppressed on incubation of syngeneic CD8+ T-cells with dendritic cells in the absence of T-cell help.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 13 OF 15 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:809019 CAPLUS
DOCUMENT NUMBER: 135:343303
TITLE: Method for enhancing an **antigen** specific immune response with **OX-40** ligand
INVENTOR(S): Weinberg, Andrew D.
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 30 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6312700	B1	20011106	US 1999-255363	19990223
US 20020054873	A1	20020509	US 2001-946832	20010904
US 7504101	B2	20090317		

US 20070207159	A1	20070906	US 2006-529956	20060929
US 7622444	B2	20091124		

PRIORITY APPLN. INFO.:

US 1998-75801P	P	19980224
US 1999-255363	A3	19990223
US 2001-946832	A1	20010904

AB Provided are compns. and methods for enhancing the immune response of a mammal to an **antigen** by engaging the **OX-40** receptor on the surface of T-cells are disclosed, comprising administering to the mammal a compn. comprising a purified **OX-40** receptor binding agent and a pharmaceutically acceptable carrier, wherein said compn. is administered to the mammal such that the **OX-40** receptor binding agent is presented to T-cells of the mammal during or shortly after priming of the T-cells by the **antigen**. Such compns. and methods can be used in immunization and cancer treatment.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 14 OF 15 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:781079 CAPLUS
DOCUMENT NUMBER: 135:348851
TITLE: Albumin fusion proteins with therapeutic proteins for improved shelf-life
INVENTOR(S): Rosen, Craig A.; Haseltine, William A.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc, USA
SOURCE: PCT Int. Appl., 606 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 90
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001079444	A2	20011025	WO 2001-US12013	20010412
WO 2001079444	A3	20020523		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001074809	A	20011020	AU 2001-74809	20010412
CA 2405557	A1	20011025	CA 2001-2405557	20010412
EP 1278544	A2	20030129	EP 2001-941457	20010412
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 20030125247	A1	20030703	US 2001-833041	20010412
US 6994857	B2	20060207		
US 20030171267	A1	20030911	US 2001-833117	20010412
JP 2003530847	T	20031021	JP 2001-577428	20010412
US 20030199043	A1	20031023	US 2001-832501	20010412
US 20030219875	A1	20031127	US 2001-833118	20010412
US 6905688	B2	20050614		

US 6946134	B1	20050920	US 2001-833111	20010412
EP 1803730	A1	20070704	EP 2006-15131	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 1832599	A2	20070912	EP 2006-76852	20010412
EP 1832599	A3	20071121		
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 1983055	A1	20081022	EP 2008-75327	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2067488	A1	20090610	EP 2008-75909	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2206720	A1	20100714	EP 2010-75064	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2213743	A1	20100804	EP 2010-75164	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2216409	A1	20100811	EP 2010-75011	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2236152	A1	20101006	EP 2010-75010	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2267026	A1	20101229	EP 2010-75387	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2275557	A1	20110119	EP 2010-75316	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2295456	A1	20110316	EP 2010-75391	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
EP 2298355	A2	20110323	EP 2010-75375	20010412
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, TR				
US 20050100991	A1	20050512	US 2004-932104	20040902
US 20070026013	A1	20070201	US 2004-933523	20040903
US 7507413	B2	20090324		
US 20050244931	A1	20051103	US 2004-967457	20041019
US 20050266532	A1	20051201	US 2005-78663	20050314
US 20070287173	A9	20071213		
US 7507414	B2	20090324		
US 20050266533	A1	20051201	US 2005-78914	20050314
US 7482013	B2	20090127		
US 20080261877	A1	20081023	US 2007-927593	20071029
US 20080267962	A1	20081030	US 2007-927555	20071029
US 20090285816	A9	20091119		
US 20080269125	A1	20081030	US 2007-927602	20071029
US 20080269126	A1	20081030	US 2007-927607	20071029
US 20080269127	A1	20081030	US 2007-927610	20071029
US 20080269128	A1	20081030	US 2007-927617	20071029
US 20080131399	A1	20080605	US 2007-929677	20071030
US 7785599	B2	20100831		
US 20090075880	A1	20090319	US 2008-140228	20080616
US 20090105140	A1	20090423	US 2008-171154	20080710
US 20100189686	A1	20100729	US 2009-365878	20090204
US 20100249026	A1	20100930	US 2010-784345	20100520

PRIORITY APPLN. INFO.:		
US 2000-229358P	P	20000412
US 2000-199384P	P	20000425
US 2000-256931P	P	20001221
EP 2001-932549	A3	20010412
EP 2001-934875	A3	20010412
EP 2001-937179	A3	20010412
EP 2006-15131	A3	20010412
EP 2006-76852	A3	20010412
EP 2008-75327	A3	20010412
EP 2008-75909	A3	20010412
US 2001-832501	B1	20010412
US 2001-833041	A3	20010412
US 2001-833111	A3	20010412
US 2001-833117	B1	20010412
US 2001-833118	A3	20010412
WO 2001-US12013	W	20010412
US 2004-932104	B1	20040902
US 2004-933523	A3	20040903
US 2004-967457	B1	20041019
US 2005-78663	A3	20050314
US 2005-78914	A3	20050314
US 2007-927492	B3	20071029

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present invention encompasses fusion proteins of albumin with various therapeutic proteins. Therapeutic proteins may be stabilized to extend the shelf-life, and/or to retain the therapeutic protein's activity for extended periods of time in soln., in vitro and/or in vivo, by genetically or chem. fusing or conjugating the therapeutic protein to albumin or a fragment or variant of albumin. Use of albumin fusion proteins may also reduce the need to formulate the protein solns. with large excesses of carrier proteins to prevent loss of therapeutic proteins due to factors such as binding to the container. Nucleic acid mols. encoding the albumin fusion proteins of the invention are also encompassed by the invention, as are vectors contg. these nucleic acids, host cells transformed with these nucleic acids vectors, and methods of making the albumin fusion proteins of the invention and using these nucleic acids, vectors, and/or host cells. Thus, **plasmid** vectors are constructed in which DNA encoding the desired therapeutic protein may be inserted for expression of the albumin fusion proteins in yeast (pPPC0005) and mammalian cells (pC4:HSA). Yeast-derived signal sequences from Saccharomyces cerevisiae invertase SUC2 gene, or the stanniocalcin or native human serum albumin signal peptides, are used for secretion in yeast or mammalian systems, resp. Thus, the fusion product of human growth hormone with residues 1-387 of human serum albumin retains essentially intact biol. activity after 5 wk of incubation in tissue culture media at 37°, whereas recombinant human growth hormone used as control lost its biol. activity in the first week. Although the potency of the albumin fusion proteins is slightly lower than the unfused counterparts in rapid bioassays, their biol. stability results in much higher biol. activity in the longer term in vitro assay or in vivo assays. Addnl., the present invention encompasses pharmaceutical compns. comprising albumin fusion proteins and methods of treating, preventing, or ameliorating diseases, disorders or conditions using albumin fusion proteins of the invention.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 15 OF 15 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2000:275313 CAPLUS

DOCUMENT NUMBER: 132:313670
TITLE: Coated substrates for blood, plasma, or tissue washing and columns equipped with these substrates
INVENTOR(S): Dunzendorfer, Udo; Will, Gottfried
PATENT ASSIGNEE(S): Germany
SOURCE: Ger. Offen., 30 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 19845286	A1	20000427	DE 1998-19845286	19981001
EP 1004598	A2	20000531	EP 1999-118541	19990918
EP 1004598	A3	20000607		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

PRIORITY APPLN. INFO.: DE 1998-19845286 A 19981001

AB Columns, filters, cannulas, etc. contg. substrates coated with specific antibodies can be used during plasmapheresis to remove pathogenic cytokines such as tumor necrosis factor (TNF), anti-TNF, fragments of TNF or anti-TNF, or TNF transport proteins from blood, plasma, or tissues. The substrates may addnl. be coated with antibodies to microbial or viral pathogens or mixts. of pathogens as well as to polysaccharide antigens, viral capsids, microbial antigens, reverse transcriptase, endothelin, protein A, etc. Selective removal of these pathogens, antigens, proteins, etc. leaves all normal plasma components unchanged and obviates the need for supplementation of the plasma with these components. Suitable substrates include polymers, polymer-coated metals, cellulose derivs., starch, and Sepharose; these may be derivatized for covalent binding of the pathogens or pathogenic mols. Thus, Escherichia coli pyelonephritis was successfully treated by plasmapheresis coupled with columns loaded with anti-TNF-α for 14 days, 4 h/day, as detd. by decreases in plasma TNF-α levels and colony counts in urine cultures.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

=> **L11 and L12**
L13 4 L11 AND L12

=> **D L13 IBIB ABS 1-4**

L13 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:103254 CAPLUS
DOCUMENT NUMBER: 144:164229
TITLE: Aminoacridine compounds for the inhibition of NF-κB, and use in the treatment of cancer and other conditions
INVENTOR(S): Gudkov, Andrei V.; Gurova, Katerina V.
PATENT ASSIGNEE(S): Cleveland Clinic Foundation, USA
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006012419	A2	20060202	WO 2005-US25884	20050720
WO 2006012419	A3	20070308		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2005267117	A1	20060202	AU 2005-267117	20050720
EP 1771203	A2	20070411	EP 2005-791579	20050720
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
JP 2008507545	T	20080313	JP 2007-522754	20050720
US 20070270455	A1	20071122	US 2007-624828	20070119
PRIORITY APPLN. INFO.:			US 2004-589637P P	20040720
			WO 2005-US25884 W	20050720

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 144:164229
AB Aminoacridines are inhibitors of NF-κB. Inhibiting NF-κB leads to reactivation of p53 in cancer cells with functionally blocked p53. The compds. of the invention are useful for the treatment of cancer and other conditions.
OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L13 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:962083 CAPLUS
DOCUMENT NUMBER: 143:246766
TITLE: Antibodies, agonists and antagonists of interleukin 27 or IL-27 receptor for diagnosis and treatment of immune disease, inflammation and cancer
INVENTOR(S): Kastelein, Robert A.; McClanahan, Terrill K.; Pflanz, Stefan
PATENT ASSIGNEE(S): Schering Corporation, USA
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005079848	A2	20050901	WO 2005-US4902	20050215
WO 2005079848	A3	20051215		
WO 2005079848	A9	20061207		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,				

CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT,
RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
MR, NE, SN, TD, TG

AU 2005215771	A1	20050901	AU 2005-215771		20050215
CA 2555421	A1	20050901	CA 2005-2555421		20050215
US 20050214296	A1	20050929	US 2005-58934		20050215
CN 1921886	A	20070228	CN 2005-80005143		20050215
EP 1755641	A2	20070228	EP 2005-713651		20050215
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU					
BR 2005007776	A	20070710	BR 2005-7776		20050215
JP 2007523169	T	20070816	JP 2006-554178		20050215
ZA 2006006833	A	20080528	ZA 2006-6833		20060816
MX 2006009438	A	20061120	MX 2006-9438		20060817
NO 2006004192	A	20061116	NO 2006-4192		20060915
PRIORITY APPLN. INFO.:			US 2004-545762P	P	20040217
			WO 2005-US4902	W	20050215

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Methods of modulating cytokine activity, e.g., for the purpose of treating immune and inflammatory disorders, are provided. Methods of administering agonists or antagonists of IL-27 and IL-27 receptor, p28, EBI3, WSX/TCCR, and WSX1/TCCR-gp130 complexes are also provided. Agonists or antagonists include antibodies, monoclonal antibodies, polyclonal antibodies, humanized antibodies, antibody fragments, peptide mimetics, or a detectable label.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:15787 CAPLUS
DOCUMENT NUMBER: 142:92172
TITLE: Cytokine-expressing cellular vaccine combinations for treatment of cancer
INVENTOR(S): Jooss, Karin; Creson, Jennifer; Li, Betty; Prell, Rodney; Aung, Sandra; Moskalenko, Marina Boris; Du, Thomas
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 52 pp., Cont.-in-part of U.S. Ser. No. 404,662.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20050002916	A1	20050106	US 2004-807449		20040324
US 20040197312	A1	20041007	US 2003-404662		20030402
PRIORITY APPLN. INFO.:			US 2003-404662	A2	20030402

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The disclosed invention provides improved methods and compns. for treating cancer in a mammal (preferably human) based on the administration of the combination of a cytokine-expressing cellular vaccine and at least one addnl. cancer therapeutic agent or treatment to a patient with cancer, wherein administration of the combination results in enhanced therapeutic efficacy relative to administration of the cytokine-expressing cellular vaccine or cancer therapeutic agent or treatment as a monotherapy. The examples present the use of cytokine-expressing cellular vaccine (GVAX) in combination with different cancer therapeutic agents in a murine melanoma model (B16F10), renal cell carcinoma (renca), colon carcinoma (CT26), breast carcinoma (4T1), lung cancer (LLC), and fibrosarcoma (3T3).

L13 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:133790 CAPLUS
DOCUMENT NUMBER: 138:168828
TITLE: Cytokine receptor-activating agent and co-stimulatory molecule-activating agent for prevention or treatment of cancer, inflammatory disorders or infectious diseases
INVENTOR(S): Chen, Shu-Hsia; Pan, Ping-Yan; Woo, Savio L. C.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 81 pp., Cont.-in-part of U.S. Ser. No. 735,296.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20030035790	A1	20030220	US 2002-165643		20020607
PRIORITY APPLN. INFO.:			US 1999-115992P	P	19990115
			US 2000-735296	A2	20000114

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The present invention relates to compns. comprising compds. which augment activated immune cells, such as T-cells, dendritic cells and natural killer ("NK") cells, and methods for the treatment or prevention of diseases and disorders, including cancer, inflammatory disorders, and infectious diseases, in a subject comprising the administration of said compns. to said subject. In particular, the present invention relates to methods for the treatment or prevention of diseases and disorders, including cancer, inflammatory disorders, and infectious diseases, in a subject comprising administrating to said subject one or more compds. that activate one or more cytokine receptors and one or more compds. that activate one or more co-stimulatory mols. expressed by activated immune cells. The present invention also relates to compns. and kits comprising a compd. that activates one or more cytokine receptors and a compd. that activates one or more co-stimulatory mols. expressed by activated immune cells.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

=> **L11 not L13**
L14 14 L11 NOT L13

=> **L12 not L13**
L15 22 L12 NOT L13

=> **D L14 IBIB ABS 1-14**

L14 ANSWER 1 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:590829 CAPLUS
DOCUMENT NUMBER: 147:29577
TITLE: Monoclonal antibody to human CD134 (OX40) for diagnosis and treatment of inflammation and autoimmune disease
INVENTOR(S): Kato, Shinichiro; Soloff Nugent, Rachel; Yoshida, Hitoshi; Croft, Michael
PATENT ASSIGNEE(S): Kirin Beer Kabushiki Kaisha, Japan; La Jolla Institute for Allergy and Immunology
SOURCE: PCT Int. Appl., 110pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007062245	A2	20070531	WO 2006-US45522	20061127
WO 2007062245	A3	20071206		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
AU 2006318329	A1	20070531	AU 2006-318329	20061127
CA 2631015	A1	20070531	CA 2006-2631015	20061127
EP 1951760	A2	20080806	EP 2006-838473	20061127
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS				
JP 2009518005	T	20090507	JP 2008-542473	20061127
KR 2008080503	A	20080904	KR 2008-7012371	20080523
CN 101331150	A	20081224	CN 2006-80044085	20080526
US 20100196359	A1	20100805	US 2009-87436	20091231
PRIORITY APPLN. INFO.:			US 2005-739659P P	20051125
			WO 2006-US45522 W	20061127

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention provides antibodies that specifically bind to OX40 (CD134), referred to as OX40 antibodies, anti-OX40 or anti-OX40 antibodies. Invention antibodies that specifically bind to OX40 include mammalian (human, primate, etc.), humanized and chimeric anti-OX40 antibodies. Invention antibodies and antibody subsequences (fragments) that specifically bind to OX40 include purified and isolated antibodies, as well as pharmaceutical formulations thereof, are useful in various methods

including treatment, screening and detection methods.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L14 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2006:499339 CAPLUS
DOCUMENT NUMBER: 145:312828
TITLE: Costimulation and autoimmune diabetes in BB rats
AUTHOR(S): Beaudette-Zlatanova, B. C.; Whalen, B.; Zipris, D.; Yagita, H.; Rozing, J.; Groen, H.; Benjamin, C. D.; Hunig, T.; Drexhage, H. A.; Ansari, M. J.; Leif, J.; Mordes, J. P.; Greiner, D. L.; Sayegh, M. H.; Rossini, A. A.
CORPORATE SOURCE: Department of Medicine, The University of Massachusetts Medical School, Worcester, MA, USA
SOURCE: American Journal of Transplantation (2006), 6(5, Pt. 1), 894-902
CODEN: AJTMBR; ISSN: 1600-6135
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Costimulatory signals regulate T-cell activation. To investigate the role of costimulation in autoimmunity and transplantation, we studied the BB rat model of type 1 diabetes. Diabetes-prone BB (BBDP) rats spontaneously develop disease when 55-120 days of age. We obsd. that two anti-CD28 monoclonal antibodies (mAb) with different functional activities completely prevented diabetes in BBDP rats. Anti-CD154 mAb delayed diabetes, whereas treatment with CTLA4-Ig or anti-CD80 mAb accelerated disease. Anti-CD86 or anti-CD134L mAbs had no effect. Diabetes resistant BB (BBDR) rats are disease-free, but >95% of them develop diabetes after treatment with polyinosinic-polycytidylic acid and an mAb that depletes Treg cells. In the induced BBDR model, anti-CD154 mAb delayed onset of diabetes, whereas CTLA4-Ig, anti-CD134L or either of the anti-CD28 mAbs had little or no effect. In contrast, blockade of the CD134-CD134L pathway was highly effective for preventing autoimmune recurrence against syngeneic islet grafts in diabetic BBDR hosts. Blockade of the CD40-CD154 pathway was also effective, but less so. These data suggest that the effectiveness of costimulation blockade in the treatment of type 1 diabetes is dependent on both the costimulatory pathway targeted and the mechanism of induction, stage, intensity and duration of the pathogenic process.
OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2006:269330 CAPLUS
DOCUMENT NUMBER: 144:348888
TITLE: Anti-OX40L antibodies for prophylaxis, diagnosis and therapy of inflammatory diseases
INVENTOR(S): Endl, Josef; Eugui, Elsie; Fuentes, Maria; Graus, Yvo; Labrijn, Aran; Lanzendoerfer, Martin; Parren, Paul; Rebers, Frank; Schumacher, Ralf; Seeber, Stefan; Van de Winkel, Jan; Van Vugt, Martine
PATENT ASSIGNEE(S): F.Hoffmann-La Roche AG, Switz.

SOURCE: PCT Int. Appl., 137 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006029879	A2	20060323	WO 2005-EP9968	20050916
WO 2006029879	A3	20060908		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
TW 309240	B	20090501	TW 2005-131726	20050914
AR 51925	A1	20070221	AR 2005-103851	20050915
AU 2005284310	A1	20060323	AU 2005-284310	20050916
CA 2580140	A1	20060323	CA 2005-2580140	20050916
EP 1791869	A2	20070606	EP 2005-791827	20050916
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
CN 101023102	A	20070822	CN 2005-80031358	20050916
JP 2008512995	T	20080501	JP 2007-531688	20050916
JP 4594986	B2	20101208		
BR 2005015554	A	20080729	BR 2005-15554	20050916
SG 147444	A1	20081128	SG 2008-7730	20050916
US 7501496	B1	20090310	US 2005-229162	20050916
NZ 553333	A	20090925	NZ 2005-553333	20050916
CN 101684157	A	20100331	CN 2009-10142533	20050916
NZ 579022	A	20100430	NZ 2005-579022	20050916
RU 2395523	C2	20100727	RU 2007-114328	20050916
EP 2218782	A2	20100818	EP 2010-155663	20050916
EP 2218782	A3	20110126		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
ZA 2007001952	A	20081029	ZA 2007-1952	20070306
MX 2007002905	A	20070508	MX 2007-2905	20070309
KR 2007050972	A	20070516	KR 2007-7006191	20070316
KR 901090	B1	20090608		
IN 2007CN01147	A	20070817	IN 2007-CN1147	20070319
IN 239940	A1	20100423		
KR 2008059471	A	20080627	KR 2008-7012656	20080527
KR 895597	B1	20090506		
US 20100166740	A1	20100701	US 2008-315863	20081205
US 7868141	B2	20110111		
IN 2009CN04675	A	20091030	IN 2009-CN4675	20090807
JP 2010280673	A	20101216	JP 2010-158015	20100712
US 20110070239	A1	20110324	US 2010-958278	20101201
PRIORITY APPLN. INFO.:			EP 2004-22158 A	20040917
			EP 2004-30546 A	20041223
			CN 2005-80031358 A3	20050916

EP 2005-791827	A3	20050916
JP 2007-531688	A3	20050916
US 2005-229162	A3	20050916
WO 2005-EP9968	W	20050916
KR 2007-7006191	A3	20070316
IN 2007-CN1147	A3	20070319
US 2008-315863	A3	20081205

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB This invention relates to anti-OX40L antibodies and, in particular, to anti-OX40L antibodies and variants thereof that contain a Fc part derived from human origin and do not bind complement factor C1q. These antibodies have new and inventive properties causing a benefit for a patient suffering from inflammatory diseases.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:164938 CAPLUS

DOCUMENT NUMBER: 145:186153

TITLE: Roles of OX40 in the pathogenesis and the control of diseases

AUTHOR(S): Hori, Toshiyuki

CORPORATE SOURCE: Department of Hematology and Oncology, Graduate School of Medicine, Kyoto University, Kyoto, Japan

SOURCE: International Journal of Hematology (2006), 83(1), 17-22
CODEN: IJHEEY; ISSN: 0925-5710

PUBLISHER: Carden Jennings Publishing

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. OX40 belongs to the tumor necrosis factor receptor superfamily, and its expression is restricted to activated T-cells. Ligation of OX40 during T-cell-dendritic cell interaction is crucial for clonal expansion of antigen-specific T-cells and generation of T-cell memory. The ligand of OX40 (OX40L) is expressed not only on dendritic cells but also on other cell types, such as B-cells, vascular endothelial cells, natural killer cells, and mast cells. The pathophysiol. relevance of this broad distribution needs further investigation. In particular, OX40L on vascular endothelial cells may play a role in inflammatory vasculitis as well as in atherosclerotic change. Recent studies with animal models have indicated the crit. involvement of OX40 in the pathogenesis of a variety of immunol. abnormalities of inflammatory, autoimmune, infectious, allergic, and allotransplantationrelated diseases. Blockade of OX40-OX40L interaction has been shown to prevent, cure, or ameliorate these diseases. In contrast, activation of OX40 is known to break an existing state of tolerance in malignancies, leading to a reactivation of antitumor immunity. These findings clearly suggest that the OX40/OX40L system is one of the most promising targets of immune intervention for treatment of these diseases.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 5 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:729611 CAPLUS
DOCUMENT NUMBER: 143:206465
TITLE: Therapeutic and carrier molecules
INVENTOR(S): Ferrante, Antonio; Rathjen, Deborah Ann
PATENT ASSIGNEE(S): Peplin Biolipids Pty Ltd, Australia
SOURCE: PCT Int. Appl., 180 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005073164	A1	20050811	WO 2005-AU98	20050128
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005209331	A1	20050811	AU 2005-209331	20050128
CA 2554735	A1	20050811	CA 2005-2554735	20050128
EP 1718602	A1	20061108	EP 2005-700130	20050128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS				
CN 1934072	A	20070321	CN 2005-80008891	20050128
BR 2005007236	A	20070626	BR 2005-7236	20050128
JP 2007522118	T	20070809	JP 2006-549788	20050128
US 20090215895	A1	20090827	US 2009-588094	20090507
PRIORITY APPLN. INFO.:			US 2004-540604P P	20040130
			WO 2005-AU98 W	20050128

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 143:206465

AB The present invention relates generally to compds. comprising a hydrocarbon chain portion and more particular to compds. comprising chem. derivatizations of the hydrocarbon chain which are useful therapeutic and prophylactic mols. The present invention further provides compds. where the hydrocarbon chain portion is a carrier mol. for functional groups, moieties or agents. The present invention can include naturally including polyunsatd. fatty acids as well as synthetic, modified or derivatized polyunsatd. fatty acids. Furthermore. these polyunsatd. fatty acids can be conjugated to amino acids, peptides or proteins. The compds. of the present invention are particularly useful in the treatment and prophylaxis of a range of conditions including cancers, protein kinase c(PKC)- or NFκB-related- or -assocd. conditions, cardiovascular conditions, pain, inflammatory conditions, vascular or immunol. conditions such as diabetes, neurol. conditions and infection by a range of viruses or prokaryotic or eukaryotic organisms. The present invention further provides pharmaceutical compns. and methods of medical treatment.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 6 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:589563 CAPLUS
DOCUMENT NUMBER: 141:134075
TITLE: Treatment of inflammation with combinations of inhibitors of interleukin 1 and of inhibitors of lymphocyte activation
INVENTOR(S): Khare, Sanjay Deep
PATENT ASSIGNEE(S): Amgen Inc., USA
SOURCE: PCT Int. Appl., 203 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2004060911	A2	20040722	WO 2003-US41378		20031224
WO 2004060911	A3	20050901			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW					
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
CA 2511823	A1	20040722	CA 2003-2511823		20031224
AU 2003299971	A1	20040729	AU 2003-299971		20031224
EP 1578782	A2	20050928	EP 2003-800236		20031224
EP 1578782	A3	20051026			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
JP 2006517191	T	20060720	JP 2004-565743		20031224
US 20040208874	A1	20041021	US 2003-748112		20031229
MX 2005007019	A	20050818	MX 2005-7019		20050627
US 20080279862	A1	20081113	US 2008-143693		20080620
PRIORITY APPLN. INFO.:			US 2002-437405P	P	20021230
			WO 2003-US41378	W	20031224
			US 2003-748112	A1	20031229

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Methods for treating inflammation, esp. in autoimmune disease, by preventing lymphocyte activation using combinations of effectors are described. These may include combinations of IL-1 inhibitors and an inhibitor of B cell or T cell activation. The use of the prior art KIN2 fusion protein of interleukin 1 receptor antagonist and Ig Fc to treat inflammation in a collagen-induced arthritis model is demonstrated. The KIN2 fusion protein lessened inflammation when used alone. A fusion protein of CTLA-4 antigen and Fc was ineffective as an inflammation inhibitor when used alone. When the two fusion proteins were used in combination, the effects on the inflammation were longer-lasting than those of KIN2 alone. Similarly, a combination of the CTLA-4 fusion protein and PEGylated tumor necrosis factor receptor 1 was more effective than the receptor alone. Use of viral vectors expressing genes for these

proteins in long-term therapy is also demonstrated.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:836393 CAPLUS
DOCUMENT NUMBER: 139:336932
TITLE: Human tumor necrosis factor-γβ, receptor DR3 and TR6, antagonists and antibodies for diagnosis, prognosis and treatment of inflammatory bowel diseases
INVENTOR(S): Yu, Guo-liang; Ni, Jian; Rosen, Craig A.; Zhang, Jun; Wei, Ping
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 173 pp., Cont.-in-part of U.S. Ser. No. 226,294.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 11
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030198640	A1	20031023	US 2002-310793	20021206
WO 9614328	A1	19960517	WO 1994-US12880	19941107
W: AU, CA, CN, JP, KR, NZ, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
JP 2002223781	A	20020813	JP 2001-337585	19941107
US 20030027284	A1	20030206	US 1998-131237	19980807
US 6599719	B2	20030729		
US 20020090683	A1	20020711	US 1999-246129	19990208
US 6824767	B2	20041130		
US 20020150534	A1	20021017	US 2001-899059	20010706
US 7597886	B2	20091006		
US 20030129189	A1	20030710	US 2002-226294	20020823

PRIORITY APPLN. INFO.:

WO 1994-US12880	A2	19941107
US 1995-461246	B2	19950605
US 1998-5020	B2	19980109
US 1998-74047P	P	19980209
US 1998-131237	A2	19980807
US 1999-246129	A2	19990208
US 1999-131963P	P	19990430
US 1999-132227P	P	19990503
US 1999-134067P	P	19990513
US 2000-180908P	P	20000208
US 2000-559290	B2	20000427
US 2000-216879P	P	20000707
US 2001-278449P	P	20010326
US 2001-899059	A2	20010706
US 2001-314381P	P	20010824
US 2001-336695P	P	20011207
US 2002-226294	A2	20020823
JP 1996-515265	A3	19941107
WO 2000-US11689	A2	20000428

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The present invention encompasses methods for detection, diagnosis,

prevention, treatment, and/or amelioration of inflammatory bowel diseases and disorders using TNF-γβ and its receptors DR3 and TR6. In particular the invention encompasses methods of using TNF-γβ, DR3 and TR6 polypeptides, as well as antibodies, and antagonists thereto, in the diagnosis, prognosis and treatment of ulcerative colitis and/or Crohn's disease. Methods of screening for antagonists of the TNF-γβ polypeptide, together with therapeutic uses of such antagonists are also disclosed.

L14 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:390790 CAPLUS
DOCUMENT NUMBER: 138:400393
TITLE: T-cell antigens, and their use in diagnosis and treatment of T-cell mediated conditions
INVENTOR(S): Weinberg, Andrew D.; Vandenbark, Arthur A.
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 29 pp., Cont.-in-part of U.S. 5,759,546.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6566082	B1	20030520	US 1995-469633	19950606
US 5759546	A	19980602	US 1994-192480	19940204
WO 9521251	A1	19950810	WO 1995-GB237	19950206
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9891341	A	19990114	AU 1998-91341	19981104
AU 9891342	A	19990121	AU 1998-91342	19981104
AU 781082	B2	20050505	AU 2001-19731	20010213
AU 782568	B2	20050811	AU 2001-79325	20011010
JP 2007045832	A	20070222	JP 2006-248393	20060913
PRIORITY APPLN. INFO.:			US 1994-192480	A2 19940204
			WO 1995-GB237	A 19950206
			AU 1995-15835	A3 19950206
			JP 1995-520474	A3 19950206
			AU 1998-91341	A3 19981104
			AU 1998-91342	A3 19981104

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The OX-40 antigen is characterized and claimed together with variants and derivs. thereof. Also described are binding agents for the antigen and the use of these in diagnosis and therapy. Examples of such use include a method for the selective depletion of activated CD4+ T-cells (without affecting the non-activated T cells) in vivo by using immunotoxins comprising an OX-40 antibody conjugated to a toxic mol. (such as ricin-A chain). The administration of these specific immunotoxins is used therapeutically to deplete autoimmune reactive CD4+ T-cells which have been implicated in diseases including multiple sclerosis, rheumatoid arthritis, sarcoidosis, and autoimmune uveitis as well as inflammatory bowel disease and graft-vs.-host disease. This type of therapy is also

beneficial for eradicating CD4+ T-cell lymphomas and alloreactive CD4+ T-cells involved with a transplantation reaction. The use of the human form of the OX-40 antibody will also help in the early diagnosis of all the diseases mentioned above.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 9 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2002:793447 CAPLUS
DOCUMENT NUMBER: 137:304813
TITLE: Modulators of hedgehog signaling pathway for treatment of T-cell-mediated diseases
INVENTOR(S): Lamb, Jonathan Robert; Hoyne, Gerard Francis; Dallman, Margaret Jane; Champion, Brian Robert
PATENT ASSIGNEE(S): Lorantis Limited, UK
SOURCE: PCT Int. Appl., 154 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002080952	A2	20021017	WO 2002-GB1666	20020409
WO 2002080952	A3	20040108		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002247847	A1	20021021	AU 2002-247847	20020409
EP 1401469	A2	20040331	EP 2002-716928	20020409
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004534743	T	20041118	JP 2002-578991	20020409
US 20040126359	A1	20040701	US 2003-682230	20031009
PRIORITY APPLN. INFO.:			GB 2001-8872 A	20010409
			GB 2001-8873 A	20010409
			WO 2002-GB1666 W	20020409

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Use of a modulator of a Hedgehog signaling pathway, or a modulator of a pathway which is a target of the Hedgehog signaling pathway in the prepn. of a medicament for treatment of a disease or disorder assocd. with a T-cell mediated disease or disorder.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 10 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:884232 CAPLUS
DOCUMENT NUMBER: 136:149820
TITLE: Augmentation versus inhibition: effects of conjunctional OX-40 receptor monoclonal antibody and IL-2 treatment on adoptive immunotherapy of advanced tumor
AUTHOR(S): Kjaergaard, Jorgen; Peng, Liaomin; Cohen, Peter A.; Drazba, Judith A.; Weinberg, Andrew D.; Shu, Suyu
CORPORATE SOURCE: Center for Surgery Research, Cleveland Clinic Foundation, Cleveland, OH, 44195, USA
SOURCE: Journal of Immunology (2001), 167(11), 6669-6677
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Therapeutic efficacy of adoptive immunotherapy of malignancies is proportional to the no. of effector T cells transferred. Traditionally, exogenous IL-2 treatment has been used to promote the survival and function of transferred cells. Recently, we described the therapeutic effects of in vivo ligation of the costimulatory receptor, OX-40R, on activated T cells during early tumor growth. In this study, we examd. the effects of IL-2 and OX-40R mAb on adoptive immunotherapy of advanced tumors. For treatment of 10-day 3-methylcholanthrene 205 pulmonary metastases, systemic transfer of 50 × 106 activated tumor-draining lymph node T cells resulted in >99% redn. of metastatic nodules. With either IL-2 or OX-40R mAb conjunctional treatment, only 20 × 106 cells were required. Advanced 10-day 3-methylcholanthrene 205 intracranial tumors could be cured by the transfer of 15 × 106 L-selectinlow T cells derived from draining lymph nodes. In this situation, IL-2 administration inhibited therapeutic effects of the transferred cells. By contrast, 5 × 106 T cells were sufficient to cure all mice if OX-40R mAb was administrated. Studies on trafficking of systemically transferred T cells revealed that IL-2, but not OX-40R mAb, impeded tumor infiltration by T cells. Tumor regression required participation of both CD4 and CD8 T cells. Because only CD4 T cells expressed OX-40R at cell transfer, direct CD4 T cell activation is possible. Alternatively, OX-40R might be up-regulated on transferred T cells at the tumor site, rendering them reactive to the mAb. Our study suggests OX-40R mAb to be a reagent of choice to augment T cell adoptive immunotherapy in clin. trials.

OS.CITING REF COUNT: 44 THERE ARE 44 CAPLUS RECORDS THAT CITE THIS RECORD (44 CITINGS)
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:777015 CAPLUS
DOCUMENT NUMBER: 135:329667
TITLE: Adult T-cell leukemia (ATL)
AUTHOR(S): Uchiyama, Takashi
CORPORATE SOURCE: Grad. Sch. Med., Kyoto Univ., Japan
SOURCE: Nippon Naika Gakkai Zasshi (2001), 90(9), 1865-1871
CODEN: NNGAAS; ISSN: 0021-5384
PUBLISHER: Nippon Naika Gakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review with refs., on recent progress in studies on the mechanism and treatment of HTLV-I (human T cell leukemia virus type I)-caused adult T-cell leukemia, discussing clin. features, in vivo proliferation mechanism of ATL cells, roles of OX40/gp34 (OX40 ligand) in in vivo proliferation of ATL cells, and therapeutic approach to ATL.

L14 ANSWER 12 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:265459 CAPLUS
DOCUMENT NUMBER: 134:290751
TITLE: Recombinant single-chain receptor antagonist proteins and their use in treatment of inflammatory disorders
INVENTOR(S): Halkier, Torben; Schambye, Hans Thalsgard; Okkels, Jens Sigurd; Andersen, Kim Vilbour; Nissen, Torben Lauesgaard; Soni, Bobby; Jeppesen, Claus Bekker; Van Den Hazel, Bart
PATENT ASSIGNEE(S): Maxygen Aps, Den.
SOURCE: PCT Int. Appl., 123 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001025277	A1	20010412	WO 2000-DK563	20001006
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1226173	A1	20020731	EP 2000-965860	20001006
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
US 20040014948	A1	20040122	US 2003-444691	20030523
PRIORITY APPLN. INFO.:			DK 1999-1438	A 19991007
			DK 1999-1855	A 19991223
			DK 2000-1119	A 20000720
			US 1999-160820P	P 19991021
			US 2000-174655P	P 20000106
			US 2000-225723P	P 20000816
			US 2000-684720	B1 20001006
			WO 2000-DK563	W 20001006

AB The invention relates to a single-chain oligomeric protein antagonist which binds to an extracellular ligand-binding domain of a cellular receptor of a type requiring binding of an oligomeric ligand to two or more receptor subunits to be activated, the protein comprising at least two, typically structurally homologous, receptor-binding sites of which at least one is capable of binding to a ligand-binding domain of the cellular receptor and at least one is incapable of effectively binding to a ligand-binding domain of the cellular receptor, whereby the single-chain oligomeric protein is capable of binding to the receptor, but incapable of activating the receptor; as well as to nucleotide sequences encoding such single-chain oligomeric proteins, expression vectors comprising such a

nucleotide sequence, recombinant host cells comprising such a nucleotide sequence or expression vector, methods for producing the nucleotide sequences and proteins, pharmaceutical compns. comprising the single-chain oligomeric protein, and use of the single-chain oligomeric protein for the prodn. of medicaments and in therapy. A preferred single-chain antagonist according to the invention is a TNF-α antagonist. Thus, a single-chain TNF-α protein comprising of 3 human TNF-α chains connected by linker peptides was produced with Saccharomyces cerevisiae and shown to be an agonist of the TNF-α receptor. The same TNF-α trimer contg. Y87R mutations in the first and third copies of TNF-α was also prepd. This was shown to be a partial TNF-α agonist and a competitive antagonist of the TNF-α receptor.

OS.CITING REF COUNT: 9 THERE ARE 9 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 13 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1999:549381 CAPLUS
DOCUMENT NUMBER: 131:156919
TITLE: Compositions containing an OX-40 receptor binding agent or a nucleic acid encoding the same and methods for enhancing antigen-specific immune response
INVENTOR(S): Weinberg, Andrew D.
PATENT ASSIGNEE(S): Sisters of Providence In Oregon, USA
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9942585	A1	19990826	WO 1999-US3908	19990223
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2321161	A1	19990826	CA 1999-2321161	19990223
AU 9928739	A	19990906	AU 1999-28739	19990223
EP 1060247	A1	20001220	EP 1999-909562	19990223
EP 1060247	B1	20080910		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2002504334	T	20020212	JP 2000-532525	19990223
AP 1261	A	20040319	AP 2000-1903	19990223
AT 408011	T	20080915	AT 1999-909562	19990223
EP 1997893	A1	20081203	EP 2008-15510	19990223
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
PT 1060247	E	20081222	PT 1999-909562	19990223
ES 2315008	T3	20090316	ES 1999-909562	19990223
MX 2000008176	A	20020311	MX 2000-8176	20000821

AU 2002302070	A1	20030320	AU 2002-302070	20021120
AU 2002302070	B2	20071115		

PRIORITY APPLN. INFO.:

US 1998-28716	A	19980224
AU 1999-28739	A3	19990223
EP 1999-909562	A3	19990223
WO 1999-US3908	W	19990223

AB Compns. and methods for enhancing the immune response of a mammal to an antigen by engaging the OX-40 receptor on the surface of T-cells are disclosed. The compn. administered to the mammal comprises a purified OX-40 receptor binding agent and a pharmaceutically acceptable carrier, wherein said compn. is administered to the mammal such that the OX-40 receptor binding agent is presented to T-cells of the mammal during or shortly after priming of the T-cells by the antigen. The OX-40 receptor binding agent may comprise an OX-40 ligand (a member of the tumor necrosis factor superfamily) or anti-OX-40 antibodies. Engagement of the OX-409 receptor on CD4+ T-cells esp. during, or shortly after, priming of such cells by antigen, can result in an increased response of the CD4+ T-cells to that antigen, and the elevated response to that antigen is maintained for a period of time substantially longer than in the absence of such an engagement. As a result of such engagement, the resistance of an animal to disease is markedly increased with improved T-cell recognition of antigens presented by infectious agents, such as bacteria and viruses, as well as tumor cells. Such compns. and methods can be used in immunization and cancer treatment. Human OX-40 ligand:Ig fusion protein is an example of a protein applicable to the present invention that can be used in human clin. trials and can stimulate human T cells.

OS.CITING REF COUNT:	3	THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)
REFERENCE COUNT:	5	THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 14 OF 14 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER:	1995:916524 CAPLUS
DOCUMENT NUMBER:	123:312224
ORIGINAL REFERENCE NO.:	123:55959a,55962a
TITLE:	**OX-40 antigen** of T lymphocyte for use in diagnosis and **treatment** of T cell-mediated conditions
INVENTOR(S):	Weinberg, Andrew Dale; Vandenbark, Arthur Allen
PATENT ASSIGNEE(S):	Cantab Pharmaceuticals Research Ltd., UK
SOURCE:	PCT Int. Appl., 92 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	2

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9521251	A1	19950810	WO 1995-GB237	19950206
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5759546	A	19980602	US 1994-192480	19940204
CA 2182685	A1	19950810	CA 1995-2182685	19950206

AU 9515835	A	19950821	AU 1995-15835	19950206
EP 742823	A1	19961120	EP 1995-907738	19950206
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09509053	T	19970916	JP 1995-520474	19950206
US 6566082	B1	20030520	US 1995-469633	19950606
AU 9891341	A	19990114	AU 1998-91341	19981104
AU 9891342	A	19990121	AU 1998-91342	19981104
AU 781082	B2	20050505	AU 2001-19731	20010213
AU 782568	B2	20050811	AU 2001-79325	20011010
JP 2007045832	A	20070222	JP 2006-248393	20060913
PRIORITY APPLN. INFO.:			US 1994-192480	A 19940204
			AU 1995-15835	A3 19950206
			JP 1995-520474	A3 19950206
			WO 1995-GB237	W 19950206
			AU 1998-91341	A3 19981104
			AU 1998-91342	A3 19981104

AB T lymphocyte **OX-40 antigen** and binding agent (e.g. antibody) for the **antigen** are provided for diagnosis and **treatment** of T cell-mediated diseases. Examples of such use include a method for the selective depletion of activated CD4+ T-cells in vivo by using immunotoxins comprising an OX-40 antibody conjugated to a cytotoxic mol. (such as Ricin-A chain). The administration of these specific immunotoxins is used therapeutically to deplete autoimmune reactive CD4+ T-cells which have been implicated in diseases including multiple sclerosis, rheumatoid arthritis, sarcoidosis, and autoimmune uveitis as well as inflammatory bowel disease and graft-vs.-host disease. This type of therapy is also beneficial for eradicating CD4+ T-cell lymphomas and alloreactive CD4+ T-cells involved with the transplantation reaction.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

=> **D L11 IBIB ABS 1-18**

L11 ANSWER 1 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:590829 CAPLUS
DOCUMENT NUMBER: 147:29577
TITLE: Monoclonal antibody to human CD134 (OX40) for diagnosis and treatment of inflammation and autoimmune disease
INVENTOR(S): Kato, Shinichiro; Soloff Nugent, Rachel; Yoshida, Hitoshi; Croft, Michael
PATENT ASSIGNEE(S): Kirin Beer Kabushiki Kaisha, Japan; La Jolla Institute for Allergy and Immunology
SOURCE: PCT Int. Appl., 110pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007062245	A2	20070531	WO 2006-US45522	20061127
WO 2007062245	A3	20071206		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN,				

KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK,
MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO,
RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT,
TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

AU 2006318329	A1	20070531	AU 2006-318329	20061127
CA 2631015	A1	20070531	CA 2006-2631015	20061127
EP 1951760	A2	20080806	EP 2006-838473	20061127
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS				
JP 2009518005	T	20090507	JP 2008-542473	20061127
KR 2008080503	A	20080904	KR 2008-7012371	20080523
CN 101331150	A	20081224	CN 2006-80044085	20080526
US 20100196359	A1	20100805	US 2009-87436	20091231
PRIORITY APPLN. INFO.:			US 2005-739659P	P 20051125
			WO 2006-US45522	W 20061127

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention provides antibodies that specifically bind to OX40 (CD134), referred to as OX40 antibodies, anti-OX40 or anti-OX40 antibodies. Invention antibodies that specifically bind to OX40 include mammalian (human, primate, etc.), humanized and chimeric anti-OX40 antibodies. Invention antibodies and antibody subsequences (fragments) that specifically bind to OX40 include purified and isolated antibodies, as well as pharmaceutical formulations thereof, are useful in various methods including treatment, screening and detection methods.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L11 ANSWER 2 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:499339 CAPLUS
DOCUMENT NUMBER: 145:312828
TITLE: Costimulation and autoimmune diabetes in BB rats
AUTHOR(S): Beaudette-Zlatanova, B. C.; Whalen, B.; Zipris, D.; Yagita, H.; Rozing, J.; Groen, H.; Benjamin, C. D.; Hunig, T.; Drexhage, H. A.; Ansari, M. J.; Leif, J.; Mordes, J. P.; Greiner, D. L.; Sayegh, M. H.; Rossini, A. A.
CORPORATE SOURCE: Department of Medicine, The University of Massachusetts Medical School, Worcester, MA, USA
SOURCE: American Journal of Transplantation (2006), 6(5, Pt. 1), 894-902
CODEN: AJTMBR; ISSN: 1600-6135
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Costimulatory signals regulate T-cell activation. To investigate the role of costimulation in autoimmunity and transplantation, we studied the BB rat model of type 1 diabetes. Diabetes-prone BB (BBDP) rats spontaneously develop disease when 55-120 days of age. We obsd. that two anti-CD28 monoclonal antibodies (mAb) with different functional activities completely prevented diabetes in BBDP rats. Anti-CD154 mAb delayed diabetes, whereas treatment with CTLA4-Ig or anti-CD80 mAb accelerated disease. Anti-CD86 or anti-CD134L mAbs had no effect. Diabetes resistant

BB (BBDR) rats are disease-free, but >95% of them develop diabetes after treatment with polyinosinic-polycytidylic acid and an mAb that depletes Treg cells. In the induced BBDR model, anti-CD154 mAb delayed onset of diabetes, whereas CTLA4-Ig, anti-CD134L or either of the anti-CD28 mAbs had little or no effect. In contrast, blockade of the CD134-CD134L pathway was highly effective for preventing autoimmune recurrence against syngeneic islet grafts in diabetic BBDR hosts. Blockade of the CD40-CD154 pathway was also effective, but less so. These data suggest that the effectiveness of costimulation blockade in the treatment of type 1 diabetes is dependent on both the costimulatory pathway targeted and the mechanism of induction, stage, intensity and duration of the pathogenic process.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:269330 CAPLUS
DOCUMENT NUMBER: 144:348888
TITLE: Anti-OX40L antibodies for prophylaxis, diagnosis and therapy of inflammatory diseases
INVENTOR(S): Endl, Josef; Eugui, Elsie; Fuentes, Maria; Graus, Yvo; Labrijn, Aran; Lanzendoerfer, Martin; Parren, Paul; Rebers, Frank; Schumacher, Ralf; Seeber, Stefan; Van de Winkel, Jan; Van Vugt, Martine
PATENT ASSIGNEE(S): F.Hoffmann-La Roche AG, Switz.
SOURCE: PCT Int. Appl., 137 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006029879	A2	20060323	WO 2005-EP9968	20050916
WO 2006029879	A3	20060908		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
TW 309240	B	20090501	TW 2005-131726	20050914
AR 51925	A1	20070221	AR 2005-103851	20050915
AU 2005284310	A1	20060323	AU 2005-284310	20050916
CA 2580140	A1	20060323	CA 2005-2580140	20050916
EP 1791869	A2	20070606	EP 2005-791827	20050916
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
CN 101023102	A	20070822	CN 2005-80031358	20050916

JP 2008512995	T	20080501	JP 2007-531688	20050916
JP 4594986	B2	20101208		
BR 2005015554	A	20080729	BR 2005-15554	20050916
SG 147444	A1	20081128	SG 2008-7730	20050916
US 7501496	B1	20090310	US 2005-229162	20050916
NZ 553333	A	20090925	NZ 2005-553333	20050916
CN 101684157	A	20100331	CN 2009-10142533	20050916
NZ 579022	A	20100430	NZ 2005-579022	20050916
RU 2395523	C2	20100727	RU 2007-114328	20050916
EP 2218782	A2	20100818	EP 2010-155663	20050916
EP 2218782	A3	20110126		
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
ZA 2007001952	A	20081029	ZA 2007-1952	20070306
MX 2007002905	A	20070508	MX 2007-2905	20070309
KR 2007050972	A	20070516	KR 2007-7006191	20070316
KR 901090	B1	20090608		
IN 2007CN01147	A	20070817	IN 2007-CN1147	20070319
IN 239940	A1	20100423		
KR 2008059471	A	20080627	KR 2008-7012656	20080527
KR 895597	B1	20090506		
US 20100166740	A1	20100701	US 2008-315863	20081205
US 7868141	B2	20110111		
IN 2009CN04675	A	20091030	IN 2009-CN4675	20090807
JP 2010280673	A	20101216	JP 2010-158015	20100712
US 20110070239	A1	20110324	US 2010-958278	20101201

PRIORITY APPLN. INFO.:	EP 2004-22158	A 20040917
	EP 2004-30546	A 20041223
	CN 2005-80031358	A3 20050916
	EP 2005-791827	A3 20050916
	JP 2007-531688	A3 20050916
	US 2005-229162	A3 20050916
	WO 2005-EP9968	W 20050916
	KR 2007-7006191	A3 20070316
	IN 2007-CN1147	A3 20070319
	US 2008-315863	A3 20081205

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB This invention relates to anti-OX40L antibodies and, in particular, to anti-OX40L antibodies and variants thereof that contain a Fc part derived from human origin and do not bind complement factor C1q. These antibodies have new and inventive properties causing a benefit for a patient suffering from inflammatory diseases.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 4 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:164938 CAPLUS
DOCUMENT NUMBER: 145:186153
TITLE: Roles of OX40 in the pathogenesis and the control of diseases
AUTHOR(S): Hori, Toshiyuki
CORPORATE SOURCE: Department of Hematology and Oncology, Graduate School of Medicine, Kyoto University, Kyoto, Japan
SOURCE: International Journal of Hematology (2006), 83(1), 17-22
CODEN: IJHEEY; ISSN: 0925-5710

PUBLISHER: Carden Jennings Publishing
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. OX40 belongs to the tumor necrosis factor receptor superfamily, and its expression is restricted to activated T-cells. Ligation of OX40 during T-cell-dendritic cell interaction is crucial for clonal expansion of antigen-specific T-cells and generation of T-cell memory. The ligand of OX40 (OX40L) is expressed not only on dendritic cells but also on other cell types, such as B-cells, vascular endothelial cells, natural killer cells, and mast cells. The pathophysiol. relevance of this broad distribution needs further investigation. In particular, OX40L on vascular endothelial cells may play a role in inflammatory vasculitis as well as in atherosclerotic change. Recent studies with animal models have indicated the crit. involvement of OX40 in the pathogenesis of a variety of immunol. abnormalities of inflammatory, autoimmune, infectious, allergic, and allotransplantationrelated diseases. Blockade of OX40-OX40L interaction has been shown to prevent, cure, or ameliorate these diseases. In contrast, activation of OX40 is known to break an existing state of tolerance in malignancies, leading to a reactivation of antitumor immunity. These findings clearly suggest that the OX40/OX40L system is one of the most promising targets of immune intervention for treatment of these diseases.
OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:103254 CAPLUS
DOCUMENT NUMBER: 144:164229
TITLE: Aminoacridine compounds for the inhibition of NF-κB, and use in the treatment of cancer and other conditions
INVENTOR(S): Gudkov, Andrei V.; Gurova, Katerina V.
PATENT ASSIGNEE(S): Cleveland Clinic Foundation, USA
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006012419	A2	20060202	WO 2005-US25884	20050720
WO 2006012419	A3	20070308		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

AU 2005267117	A1	20060202	AU 2005-267117	20050720
EP 1771203	A2	20070411	EP 2005-791579	20050720
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
JP 2008507545	T	20080313	JP 2007-522754	20050720
US 20070270455	A1	20071122	US 2007-624828	20070119
PRIORITY APPLN. INFO.:			US 2004-589637P	P 20040720
			WO 2005-US25884	W 20050720

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 144:164229
AB Aminoacridines are inhibitors of NF-κB. Inhibiting NF-κB leads to reactivation of p53 in cancer cells with functionally blocked p53. The compds. of the invention are useful for the treatment of cancer and other conditions.
OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L11 ANSWER 6 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:962083 CAPLUS
DOCUMENT NUMBER: 143:246766
TITLE: Antibodies, agonists and antagonists of interleukin 27 or IL-27 receptor for diagnosis and treatment of immune disease, inflammation and cancer
INVENTOR(S): Kastelein, Robert A.; McClanahan, Terrill K.; Pflanz, Stefan
PATENT ASSIGNEE(S): Schering Corporation, USA
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005079848	A2	20050901	WO 2005-US4902	20050215
WO 2005079848	A3	20051215		
WO 2005079848	A9	20061207		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005215771	A1	20050901	AU 2005-215771	20050215
CA 2555421	A1	20050901	CA 2005-2555421	20050215
US 20050214296	A1	20050929	US 2005-58934	20050215
CN 1921886	A	20070228	CN 2005-80005143	20050215
EP 1755641	A2	20070228	EP 2005-713651	20050215
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, LV, MK, YU				

BR 2005007776	A	20070710	BR 2005-7776	20050215
JP 2007523169	T	20070816	JP 2006-554178	20050215
ZA 2006006833	A	20080528	ZA 2006-6833	20060816
MX 2006009438	A	20061120	MX 2006-9438	20060817
NO 2006004192	A	20061116	NO 2006-4192	20060915
PRIORITY APPLN. INFO.:			US 2004-545762P P	20040217
			WO 2005-US4902 W	20050215

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Methods of modulating cytokine activity, e.g., for the purpose of treating immune and inflammatory disorders, are provided. Methods of administering agonists or antagonists of IL-27 and IL-27 receptor, p28, EBI3, WSX/TCCR, and WSX1/TCCR-gp130 complexes are also provided. Agonists or antagonists include antibodies, monoclonal antibodies, polyclonal antibodies, humanized antibodies, antibody fragments, peptide mimetics, or a detectable label.

REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 7 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:729611 CAPLUS
DOCUMENT NUMBER: 143:206465
TITLE: Therapeutic and carrier molecules
INVENTOR(S): Ferrante, Antonio; Rathjen, Deborah Ann
PATENT ASSIGNEE(S): Peplin Biolipids Pty Ltd, Australia
SOURCE: PCT Int. Appl., 180 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005073164	A1	20050811	WO 2005-AU98	20050128
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2005209331	A1	20050811	AU 2005-209331	20050128
CA 2554735	A1	20050811	CA 2005-2554735	20050128
EP 1718602	A1	20061108	EP 2005-700130	20050128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS				
CN 1934072	A	20070321	CN 2005-80008891	20050128
BR 2005007236	A	20070626	BR 2005-7236	20050128
JP 2007522118	T	20070809	JP 2006-549788	20050128
US 20090215895	A1	20090827	US 2009-588094	20090507
PRIORITY APPLN. INFO.:			US 2004-540604P P	20040130
			WO 2005-AU98 W	20050128

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 143:206465

AB The present invention relates generally to compds. comprising a hydrocarbon chain portion and more particular to compds. comprising chem. derivatizations of the hydrocarbon chain which are useful therapeutic and prophylactic mols. The present invention further provides compds. where the hydrocarbon chain portion is a carrier mol. for functional groups, moieties or agents. The present invention can include naturally including polyunsatd. fatty acids as well as synthetic, modified or derivatized polyunsatd. fatty acids. Furthermore. these polyunsatd. fatty acids can be conjugated to amino acids, peptides or proteins. The compds. of the present invention are particularly useful in the treatment and prophylaxis of a range of conditions including cancers, protein kinase c(PKC)- or NFκB-related- or -assocd. conditions, cardiovascular conditions, pain, inflammatory conditions, vascular or immunol. conditions such as diabetes, neurol. conditions and infection by a range of viruses or prokaryotic or eukaryotic organisms. The present invention further provides pharmaceutical compns. and methods of medical treatment.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:15787 CAPLUS
DOCUMENT NUMBER: 142:92172
TITLE: Cytokine-expressing cellular vaccine combinations for treatment of cancer
INVENTOR(S): Jooss, Karin; Creson, Jennifer; Li, Betty; Prell, Rodney; Aung, Sandra; Moskalenko, Marina Boris; Du, Thomas
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 52 pp., Cont.-in-part of U.S. Ser. No. 404,662.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20050002916	A1	20050106	US 2004-807449	20040324
US 20040197312	A1	20041007	US 2003-404662	20030402
PRIORITY APPLN. INFO.:			US 2003-404662 A2	20030402

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The disclosed invention provides improved methods and compns. for treating cancer in a mammal (preferably human) based on the administration of the combination of a cytokine-expressing cellular vaccine and at least one addnl. cancer therapeutic agent or treatment to a patient with cancer, wherein administration of the combination results in enhanced therapeutic efficacy relative to administration of the cytokine-expressing cellular vaccine or cancer therapeutic agent or treatment as a monotherapy. The examples present the use of cytokine-expressing cellular vaccine (GVAX) in combination with different cancer therapeutic agents in a murine melanoma model (B16F10), renal cell carcinoma (renca), colon carcinoma (CT26), breast carcinoma (4T1), lung cancer (LLC), and fibrosarcoma (3T3).

L11 ANSWER 9 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:589563 CAPLUS
DOCUMENT NUMBER: 141:134075
TITLE: Treatment of inflammation with combinations of inhibitors of interleukin 1 and of inhibitors of lymphocyte activation
INVENTOR(S): Khare, Sanjay Deep
PATENT ASSIGNEE(S): Amgen Inc., USA
SOURCE: PCT Int. Appl., 203 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004060911	A2	20040722	WO 2003-US41378	20031224
WO 2004060911	A3	20050901		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2511823	A1	20040722	CA 2003-2511823	20031224
AU 2003299971	A1	20040729	AU 2003-299971	20031224
EP 1578782	A2	20050928	EP 2003-800236	20031224
EP 1578782	A3	20051026		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006517191	T	20060720	JP 2004-565743	20031224
US 20040208874	A1	20041021	US 2003-748112	20031229
MX 2005007019	A	20050818	MX 2005-7019	20050627
US 20080279862	A1	20081113	US 2008-143693	20080620
PRIORITY APPLN. INFO.:			US 2002-437405P	P 20021230
			WO 2003-US41378	W 20031224
			US 2003-748112	A1 20031229

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Methods for treating inflammation, esp. in autoimmune disease, by preventing lymphocyte activation using combinations of effectors are described. These may include combinations of IL-1 inhibitors and an inhibitor of B cell or T cell activation. The use of the prior art KIN2 fusion protein of interleukin 1 receptor antagonist and Ig Fc to treat inflammation in a collagen-induced arthritis model is demonstrated. The KIN2 fusion protein lessened inflammation when used alone. A fusion protein of CTLA-4 antigen and Fc was ineffective as an inflammation inhibitor when used alone. When the two fusion proteins were used in combination, the effects on the inflammation were longer-lasting than those of KIN2 alone. Similarly, a combination of the CTLA-4 fusion protein and PEGylated tumor necrosis factor receptor 1 was more effective than the receptor alone. Use of viral vectors expressing genes for these proteins in long-term therapy is also demonstrated.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 10 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:836393 CAPLUS
DOCUMENT NUMBER: 139:336932
TITLE: Human tumor necrosis factor-γβ, receptor DR3 and TR6, antagonists and antibodies for diagnosis, prognosis and treatment of inflammatory bowel diseases
INVENTOR(S): Yu, Guo-liang; Ni, Jian; Rosen, Craig A.; Zhang, Jun; Wei, Ping
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 173 pp., Cont.-in-part of U.S. Ser. No. 226,294.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 11
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030198640	A1	20031023	US 2002-310793	20021206
WO 9614328	A1	19960517	WO 1994-US12880	19941107
W: AU, CA, CN, JP, KR, NZ, US				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
JP 2002223781	A	20020813	JP 2001-337585	19941107
US 20030027284	A1	20030206	US 1998-131237	19980807
US 6599719	B2	20030729		
US 20020090683	A1	20020711	US 1999-246129	19990208
US 6824767	B2	20041130		
US 20020150534	A1	20021017	US 2001-899059	20010706
US 7597886	B2	20091006		
US 20030129189	A1	20030710	US 2002-226294	20020823

PRIORITY APPLN. INFO.:

WO 1994-US12880	A2	19941107
US 1995-461246	B2	19950605
US 1998-5020	B2	19980109
US 1998-74047P	P	19980209
US 1998-131237	A2	19980807
US 1999-246129	A2	19990208
US 1999-131963P	P	19990430
US 1999-132227P	P	19990503
US 1999-134067P	P	19990513
US 2000-180908P	P	20000208
US 2000-559290	B2	20000427
US 2000-216879P	P	20000707
US 2001-278449P	P	20010326
US 2001-899059	A2	20010706
US 2001-314381P	P	20010824
US 2001-336695P	P	20011207
US 2002-226294	A2	20020823
JP 1996-515265	A3	19941107
WO 2000-US11689	A2	20000428

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The present invention encompasses methods for detection, diagnosis, prevention, treatment, and/or amelioration of inflammatory bowel diseases and disorders using TNF-γβ and its receptors DR3 and TR6. In particular the invention encompasses methods of using TNF-γβ,

DR3 and TR6 polypeptides, as well as antibodies, and antagonists thereto, in the diagnosis, prognosis and treatment of ulcerative colitis and/or Crohn's disease. Methods of screening for antagonists of the TNF-γβ polypeptide, together with therapeutic uses of such antagonists are also disclosed.

L11 ANSWER 11 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2003:390790 CAPLUS
DOCUMENT NUMBER: 138:400393
TITLE: T-cell antigens, and their use in diagnosis and treatment of T-cell mediated conditions
INVENTOR(S): Weinberg, Andrew D.; Vandenbark, Arthur A.
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 29 pp., Cont.-in-part of U.S. 5,759,546. CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6566082	B1	20030520	US 1995-469633	19950606
US 5759546	A	19980602	US 1994-192480	19940204
WO 9521251	A1	19950810	WO 1995-GB237	19950206
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9891341	A	19990114	AU 1998-91341	19981104
AU 9891342	A	19990121	AU 1998-91342	19981104
AU 781082	B2	20050505	AU 2001-19731	20010213
AU 782568	B2	20050811	AU 2001-79325	20011010
JP 2007045832	A	20070222	JP 2006-248393	20060913

PRIORITY APPLN. INFO.:

US 1994-192480	A2	19940204
WO 1995-GB237	A	19950206
AU 1995-15835	A3	19950206
JP 1995-520474	A3	19950206
AU 1998-91341	A3	19981104
AU 1998-91342	A3	19981104

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The OX-40 antigen is characterized and claimed together with variants and derivs. thereof. Also described are binding agents for the antigen and the use of these in diagnosis and therapy. Examples of such use include a method for the selective depletion of activated CD4+ T-cells (without affecting the non-activated T cells) in vivo by using immunotoxins comprising an OX-40 antibody conjugated to a toxic mol. (such as ricin-A chain). The administration of these specific immunotoxins is used therapeutically to deplete autoimmune reactive CD4+ T-cells which have been implicated in diseases including multiple sclerosis, rheumatoid arthritis, sarcoidosis, and autoimmune uveitis as well as inflammatory bowel disease and graft-vs.-host disease. This type of therapy is also beneficial for eradicating CD4+ T-cell lymphomas and alloreactive CD4+ T-cells involved with a transplantation reaction. The use of the human form of the OX-40 antibody will also help in the early diagnosis of all

the diseases mentioned above.
OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 12 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2003:133790 CAPLUS
DOCUMENT NUMBER: 138:168828
TITLE: Cytokine receptor-activating agent and co-stimulatory molecule-activating agent for prevention or treatment of cancer, inflammatory disorders or infectious diseases
INVENTOR(S): Chen, Shu-Hsia; Pan, Ping-Yan; Woo, Savio L. C.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 81 pp., Cont.-in-part of U.S. Ser. No. 735,296.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 20030035790	A1	20030220	US 2002-165643		20020607
PRIORITY APPLN. INFO.:			US 1999-115992P	P	19990115
			US 2000-735296	A2	20000114

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The present invention relates to compns. comprising compds. which augment activated immune cells, such as T-cells, dendritic cells and natural killer ("NK") cells, and methods for the treatment or prevention of diseases and disorders, including cancer, inflammatory disorders, and infectious diseases, in a subject comprising the administration of said compns. to said subject. In particular, the present invention relates to methods for the treatment or prevention of diseases and disorders, including cancer, inflammatory disorders, and infectious diseases, in a subject comprising administrating to said subject one or more compds. that activate one or more cytokine receptors and one or more compds. that activate one or more co-stimulatory mols. expressed by activated immune cells. The present invention also relates to compns. and kits comprising a compd. that activates one or more cytokine receptors and a compd. that activates one or more co-stimulatory mols. expressed by activated immune cells.
OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L11 ANSWER 13 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2002:793447 CAPLUS
DOCUMENT NUMBER: 137:304813
TITLE: Modulators of hedgehog signaling pathway for treatment of T-cell-mediated diseases
INVENTOR(S): Lamb, Jonathan Robert; Hoyne, Gerard Francis; Dallman, Margaret Jane; Champion, Brian Robert
PATENT ASSIGNEE(S): Lorantis Limited, UK
SOURCE: PCT Int. Appl., 154 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002080952	A2	20021017	WO 2002-GB1666	20020409
WO 2002080952	A3	20040108		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002247847	A1	20021021	AU 2002-247847	20020409
EP 1401469	A2	20040331	EP 2002-716928	20020409
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
JP 2004534743	T	20041118	JP 2002-578991	20020409
US 20040126359	A1	20040701	US 2003-682230	20031009
PRIORITY APPLN. INFO.:			GB 2001-8872 A	20010409
			GB 2001-8873 A	20010409
			WO 2002-GB1666 W	20020409

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Use of a modulator of a Hedgehog signaling pathway, or a modulator of a pathway which is a target of the Hedgehog signaling pathway in the prepn. of a medicament for treatment of a disease or disorder assocd. with a T-cell mediated disease or disorder.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 14 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:884232 CAPLUS
DOCUMENT NUMBER: 136:149820
TITLE: Augmentation versus inhibition: effects of conjunctional OX-40 receptor monoclonal antibody and IL-2 treatment on adoptive immunotherapy of advanced tumor
AUTHOR(S): Kjaergaard, Jorgen; Peng, Liaomin; Cohen, Peter A.; Drazba, Judith A.; Weinberg, Andrew D.; Shu, Suyu
CORPORATE SOURCE: Center for Surgery Research, Cleveland Clinic Foundation, Cleveland, OH, 44195, USA
SOURCE: Journal of Immunology (2001), 167(11), 6669-6677
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Therapeutic efficacy of adoptive immunotherapy of malignancies is proportional to the no. of effector T cells transferred. Traditionally, exogenous IL-2 treatment has been used to promote the survival and

function of transferred cells. Recently, we described the therapeutic effects of in vivo ligation of the costimulatory receptor, OX-40R, on activated T cells during early tumor growth. In this study, we examd. the effects of IL-2 and OX-40R mAb on adoptive immunotherapy of advanced tumors. For treatment of 10-day 3-methylcholanthrene 205 pulmonary metastases, systemic transfer of 50 × 106 activated tumor-draining lymph node T cells resulted in >99% redn. of metastatic nodules. With either IL-2 or OX-40R mAb conjunctional treatment, only 20 × 106 cells were required. Advanced 10-day 3-methylcholanthrene 205 intracranial tumors could be cured by the transfer of 15 × 106 L-selectinlow T cells derived from draining lymph nodes. In this situation, IL-2 administration inhibited therapeutic effects of the transferred cells. By contrast, 5 × 106 T cells were sufficient to cure all mice if OX-40R mAb was administrated. Studies on trafficking of systemically transferred T cells revealed that IL-2, but not OX-40R mAb, impeded tumor infiltration by T cells. Tumor regression required participation of both CD4 and CD8 T cells. Because only CD4 T cells expressed OX-40R at cell transfer, direct CD4 T cell activation is possible. Alternatively, OX-40R might be up-regulated on transferred T cells at the tumor site, rendering them reactive to the mAb. Our study suggests OX-40R mAb to be a reagent of choice to augment T cell adoptive immunotherapy in clin. trials.

OS.CITING REF COUNT: 44 THERE ARE 44 CAPLUS RECORDS THAT CITE THIS RECORD (44 CITINGS)

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 15 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:777015 CAPLUS
DOCUMENT NUMBER: 135:329667
TITLE: Adult T-cell leukemia (ATL)
AUTHOR(S): Uchiyama, Takashi
CORPORATE SOURCE: Grad. Sch. Med., Kyoto Univ., Japan
SOURCE: Nippon Naika Gakkai Zasshi (2001), 90(9), 1865-1871
CODEN: NNGAAS; ISSN: 0021-5384
PUBLISHER: Nippon Naika Gakkai
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

AB A review with refs., on recent progress in studies on the mechanism and treatment of HTLV-I (human T cell leukemia virus type I)-caused adult T-cell leukemia, discussing clin. features, in vivo proliferation mechanism of ATL cells, roles of OX40/gp34 (OX40 ligand) in in vivo proliferation of ATL cells, and therapeutic approach to ATL.

L11 ANSWER 16 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2001:265459 CAPLUS
DOCUMENT NUMBER: 134:290751
TITLE: Recombinant single-chain receptor antagonist proteins and their use in treatment of inflammatory disorders
INVENTOR(S): Halkier, Torben; Schambye, Hans Thalsgard; Okkels, Jens Sigurd; Andersen, Kim Vilbour; Nissen, Torben Lauesgaard; Soni, Bobby; Jeppesen, Claus Bekker; Van Den Hazel, Bart
PATENT ASSIGNEE(S): Maxygen Aps, Den.
SOURCE: PCT Int. Appl., 123 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001025277	A1	20010412	WO 2000-DK563	20001006
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1226173	A1	20020731	EP 2000-965860	20001006
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
US 20040014948	A1	20040122	US 2003-444691	20030523

PRIORITY APPLN. INFO.:			
DK 1999-1438	A	19991007
DK 1999-1855	A	19991223
DK 2000-1119	A	20000720
US 1999-160820P	P	19991021
US 2000-174655P	P	20000106
US 2000-225723P	P	20000816
US 2000-684720	B1	20001006
WO 2000-DK563	W	20001006

AB The invention relates to a single-chain oligomeric protein antagonist which binds to an extracellular ligand-binding domain of a cellular receptor of a type requiring binding of an oligomeric ligand to two or more receptor subunits to be activated, the protein comprising at least two, typically structurally homologous, receptor-binding sites of which at least one is capable of binding to a ligand-binding domain of the cellular receptor and at least one is incapable of effectively binding to a ligand-binding domain of the cellular receptor, whereby the single-chain oligomeric protein is capable of binding to the receptor, but incapable of activating the receptor; as well as to nucleotide sequences encoding such single-chain oligomeric proteins, expression vectors comprising such a nucleotide sequence, recombinant host cells comprising such a nucleotide sequence or expression vector, methods for producing the nucleotide sequences and proteins, pharmaceutical compns. comprising the single-chain oligomeric protein, and use of the single-chain oligomeric protein for the prodn. of medicaments and in therapy. A preferred single-chain antagonist according to the invention is a TNF-α antagonist. Thus, a single-chain TNF-α protein comprising of 3 human TNF-α chains connected by linker peptides was produced with Saccharomyces cerevisiae and shown to be an agonist of the TNF-α receptor. The same TNF-α trimer contg. Y87R mutations in the first and third copies of TNF-α was also prepd. This was shown to be a partial TNF-α agonist and a competitive antagonist of the TNF-α receptor.

OS.CITING REF COUNT: 9 THERE ARE 9 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)
REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 17 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1999:549381 CAPLUS
DOCUMENT NUMBER: 131:156919
TITLE: Compositions containing an OX-40 receptor binding agent or a nucleic acid encoding the same and methods for enhancing antigen-specific immune response
INVENTOR(S): Weinberg, Andrew D.
PATENT ASSIGNEE(S): Sisters of Providence In Oregon, USA
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9942585	A1	19990826	WO 1999-US3908	19990223
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2321161	A1	19990826	CA 1999-2321161	19990223
AU 9928739	A	19990906	AU 1999-28739	19990223
EP 1060247	A1	20001220	EP 1999-909562	19990223
EP 1060247	B1	20080910		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2002504334	T	20020212	JP 2000-532525	19990223
AP 1261	A	20040319	AP 2000-1903	19990223
AT 408011	T	20080915	AT 1999-909562	19990223
EP 1997893	A1	20081203	EP 2008-15510	19990223
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
PT 1060247	E	20081222	PT 1999-909562	19990223
ES 2315008	T3	20090316	ES 1999-909562	19990223
MX 2000008176	A	20020311	MX 2000-8176	20000821
AU 2002302070	A1	20030320	AU 2002-302070	20021120
AU 2002302070	B2	20071115		

PRIORITY APPLN. INFO.:		
US 1998-28716	A	19980224
AU 1999-28739	A3	19990223
EP 1999-909562	A3	19990223
WO 1999-US3908	W	19990223

AB Compns. and methods for enhancing the immune response of a mammal to an antigen by engaging the OX-40 receptor on the surface of T-cells are disclosed. The compn. administered to the mammal comprises a purified OX-40 receptor binding agent and a pharmaceutically acceptable carrier, wherein said compn. is administered to the mammal such that the OX-40 receptor binding agent is presented to T-cells of the mammal during or shortly after priming of the T-cells by the antigen. The OX-40 receptor binding agent may comprise an OX-40 ligand (a member of the tumor necrosis factor superfamily) or anti-OX-40 antibodies. Engagement of the OX-409 receptor on CD4+ T-cells esp. during, or shortly after, priming of such cells by antigen, can result in an increased response of the CD4+ T-cells to that antigen, and the elevated response to that antigen is maintained for a period of time substantially longer than in the absence of such an engagement. As a result of such engagement, the resistance of an animal

to disease is markedly increased with improved T-cell recognition of antigens presented by infectious agents, such as bacteria and viruses, as well as tumor cells. Such compns. and methods can be used in immunization and cancer treatment. Human OX-40 ligand:Ig fusion protein is an example of a protein applicable to the present invention that can be used in human clin. trials and can stimulate human T cells.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 18 OF 18 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 1995:916524 CAPLUS
DOCUMENT NUMBER: 123:312224
ORIGINAL REFERENCE NO.: 123:55959a,55962a
TITLE: **OX-40 antigen** of T lymphocyte for use in diagnosis and **treatment** of T cell-mediated conditions
INVENTOR(S): Weinberg, Andrew Dale; Vandenbark, Arthur Allen
PATENT ASSIGNEE(S): Cantab Pharmaceuticals Research Ltd., UK
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9521251	A1	19950810	WO 1995-GB237	19950206
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NL, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US				
RW: KE, MW, SD, SZ, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5759546	A	19980602	US 1994-192480	19940204
CA 2182685	A1	19950810	CA 1995-2182685	19950206
AU 9515835	A	19950821	AU 1995-15835	19950206
EP 742823	A1	19961120	EP 1995-907738	19950206
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 09509053	T	19970916	JP 1995-520474	19950206
US 6566082	B1	20030520	US 1995-469633	19950606
AU 9891341	A	19990114	AU 1998-91341	19981104
AU 9891342	A	19990121	AU 1998-91342	19981104
AU 781082	B2	20050505	AU 2001-19731	20010213
AU 782568	B2	20050811	AU 2001-79325	20011010
JP 2007045832	A	20070222	JP 2006-248393	20060913

PRIORITY APPLN. INFO.:		
US 1994-192480	A	19940204
AU 1995-15835	A3	19950206
JP 1995-520474	A3	19950206
WO 1995-GB237	W	19950206
AU 1998-91341	A3	19981104
AU 1998-91342	A3	19981104

AB T lymphocyte **OX-40 antigen** and binding agent (e.g. antibody) for the **antigen** are provided for diagnosis and **treatment** of T cell-mediated diseases. Examples of such use include a method for the selective depletion of activated CD4+ T-cells in vivo by using immunotoxins

comprising an OX-40 antibody conjugated to a cytotoxic mol. (such as Ricin-A chain). The administration of these specific immunotoxins is used therapeutically to deplete autoimmune reactive CD4+ T-cells which have been implicated in diseases including multiple sclerosis, rheumatoid arthritis, sarcoidosis, and autoimmune uveitis as well as inflammatory bowel disease and graft-vs.-host disease. This type of therapy is also beneficial for eradicating CD4+ T-cell lymphomas and alloreactive CD4+ T-cells involved with the transplantation reaction.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

=> **immunogen same Fos**
L16 0 IMMUNOGEN SAME FOS

=> **immunogen (s) fos**
L17 1 IMMUNOGEN (S) FOS

=> **DNA vaccine (s) fos**
L18 7 DNA VACCINE (S) FOS

=> **Trial (s) DNA vaccine**
L19 395 TRIAL (S) DNA VACCINE

=> **antigen (s) L19**
L20 23 ANTIGEN (S) L19

=> **D L18 IBIB ABS 1-7**

L18 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:347184 CAPLUS
DOCUMENT NUMBER: 142:409700
TITLE: DNA vaccines against tumor growth
INVENTOR(S): Luo, Yunping; Xiang, Rong; Reisfeld, Ralph A.
PATENT ASSIGNEE(S): The Scripps Research Institute, USA
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005035777	A1	20050421	WO 2004-US33137	20041007
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 20070110717	A1	20070517	US 2006-574752	20060406
US 7569552	B2	20090804		

US 20100136058	A1	20100603	US 2009-462496		20090804
PRIORITY APPLN. INFO.:			US 2003-509457P	P	20031008
			WO 2004-US33137	W	20041007
			US 2006-574752	A1	20060406

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The authors disclose DNA vaccines suitable for eliciting an immune response against cancer cells. Th vaccines comprise a polynucleotide construct operably encoding an a Fra-1 protein, such as a polyubiquitinated human Fra-1 protein, and IL-18, such as human IL-18, in a pharmaceutically acceptable carrier. In a preferred embodiment, the polynucleotide construct is operably incorporated in an attenuated bacterial vector, such as an attenuated Salmonella typhimurium, particularly a doubly attenuated aroA- dam- S. typhimurium. Transformed host cells, methods of inhibiting tumor growth, of vaccinating a patient against cancer, and of delivering genetic material to a mammalian cell in vivo are also described.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:328835 CAPLUS
DOCUMENT NUMBER: 142:409367
TITLE: A **DNA Vaccine** Targeting **Fos**-Related Antigen 1 Enhanced by IL-18 Induces Long-lived T-Cell Memory against Tumor Recurrence
AUTHOR(S): Luo, Yunping; Zhou, He; Mizutani, Masato; Mizutani, Noriko; Liu, Cheng; Xiang, Rong; Reisfeld, Ralph A.
CORPORATE SOURCE: Department of Immunology, Scripps Research Institute, La Jolla, CA, USA
SOURCE: Cancer Research (2005), 65(8), 3419-3427
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A novel vaccination strategy induced specific CD8+ T cell-mediated immunity that eradicated spontaneous and exptl. pulmonary cancer metastases in syngeneic mice and was also effective in a therapeutic setting of established breast cancer metastases. This was achieved by targeting transcription factor **Fos**-related antigen 1(Fra-1), overexpressed by many tumor cells, with an ubiquitinated **DNA vaccine** against Fra-1, coexpressing secretory IL-18. Insight into the immunol. mechanisms involved was provided by adoptive transfer of T lymphocytes from successfully immunized BALB/c mice to syngeneic severe combined immunodeficient (SCID) mice. Specifically, long-lived T memory cells were maintained dormant in nonlymphoid tissues by IL-18 in the absence of tumor antigen. Importantly, a second tumor cell challenge of these SCID mice restored both, robust tumor-specific cytotoxicity and long-lived T-cell memory, capable of eradicating established pulmonary cancer metastases, suggesting that this vaccine could be effective against tumor recurrence.

OS.CITING REF COUNT: 24 THERE ARE 24 CAPLUS RECORDS THAT CITE THIS RECORD (24 CITINGS)
REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:1022267 CAPLUS

DOCUMENT NUMBER: 142:238041
TITLE: DNA Vaccines Suppress Angiogenesis and Protect Against Growth of Breast Cancer Metastases
AUTHOR(S): Mizutani, N.; Luo, Y.; Mizutani, M.; Reisfeld, R. A.; Xiang, R.
CORPORATE SOURCE: The Scripps Research Institute, La Jolla, CA, 92037, USA
SOURCE: Breast Disease (2004), 20, 81-91
CODEN: BRDIE5; ISSN: 0888-6008
PUBLISHER: IOS Press
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Two novel oral DNA-based vaccines provide immune protection against breast cancer in mouse model systems. These vaccines are delivered by attenuated Salmonella typhimurium to secondary lymphoid organs and are directed against novel targets such as transcription factor Fos-related antigen 1 (Fra-1) and endoglin (CD105). Both vaccines elicit suppression of angiogenesis in the breast tumor vasculature and break peripheral tolerance by eliciting potent cell-mediated protective immunity against these tumor self-antigens resulting in effective suppression of breast tumor growth and metastasis.
OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2004:286197 CAPLUS
DOCUMENT NUMBER: 141:138646
TITLE: DNA Vaccines Designed to Inhibit Tumor Growth by Suppression of Angiogenesis
AUTHOR(S): Reisfeld, Ralph A.; Niethammer, A. G.; Luo, Y.; Xiang, R.
CORPORATE SOURCE: The Scripps Research Institute, La Jolla, CA, USA
SOURCE: International Archives of Allergy and Immunology (2004), 133(3), 295-304
CODEN: IAAIEG; ISSN: 1018-2438
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The development of new blood vessels, i.e. angiogenesis, is a rate-limiting step in the development of tumors since tumor growth is generally limited to 1-2 mm3 in the absence of a blood supply. Thus, the inhibition of tumor growth by attacking the tumor's vascular supply offers a primary target for antiangiogenic intervention by DNA-based vaccines. Here, we describe two novel orally delivered DNA vaccines which suppress tumor angiogenesis and induce a robust cell-mediated immune response that provides for long-lived protection against melanoma, colon, breast and non-small-cell lung carcinoma in mouse model systems. These vaccines, which are delivered by attenuated Salmonella typhimurium to secondary lymphoid organs, are directed against such targets as vascular endothelial growth factor receptor 2 (FLK-1) and transcription factor Fos-related antigen 1 (Fra-1). Both vaccines break peripheral T cell tolerance against these self-antigens and induce a robust T cell-mediated immune response leading to suppression of tumor angiogenesis and resulting in effective suppression of tumor growth and metastases. Such research efforts may open up new possibilities for the rational design of future DNA vaccines effective for the prevention and treatment of cancer.

OS.CITING REF COUNT: 14 THERE ARE 14 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)
REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2003:588164 CAPLUS
DOCUMENT NUMBER: 139:178433
TITLE: Transcription factor Fos-related antigen 1 is an effective target for a breast cancer vaccine
AUTHOR(S): Luo, Yunping; Zhou, He; Mizutani, Masato; Mizutani, Noriko; Reisfeld, Ralph A.; Xiang, Rong
CORPORATE SOURCE: Department of Immunology, The Scripps Research Institute, La Jolla, CA, 92037, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2003), 100(15), 8850-8855
CODEN: PNASA6; ISSN: 0027-8424
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Protection against breast cancer was achieved with a **DNA vaccine** against murine transcription factor **Fos**-related antigen 1, which is overexpressed in aggressively proliferating D2F2 murine breast carcinoma. Growth of primary s.c. tumor and dissemination of pulmonary metastases was markedly suppressed by this oral **DNA vaccine**, carried by attenuated Salmonella typhimurium, encoding murine **Fos**-related antigen 1, fused with mutant polyubiquitin, and contransformed with secretory murine IL-18. The life span of 60% of vaccinated mice was tripled in the absence of detectable tumor growth after lethal tumor cell challenge. Immunol. mechanisms involved activation of T, natural killer, and dendritic cells, as indicated by up-regulation of their activation markers and costimulatory mols. Markedly increased specific target cell lysis was mediated by both MHC class I-restricted CD8+ T cells and natural killer cells isolated from splenocytes of vaccinated mice, including a significant release of proinflammatory cytokines IFN-γ and IL-2. Importantly, fluorescence anal. of fibroblast growth factor 2 and tumor cell-induced vessel growth in Matrigel plugs demonstrated marked suppression of angiogenesis only in vaccinated animals. Taken together, this multifunctional DNA vaccine proved effective in protecting against growth and metastases of breast cancer by combining the action of immune effector cells with suppression of tumor angiogenesis.

OS.CITING REF COUNT: 50 THERE ARE 50 CAPLUS RECORDS THAT CITE THIS RECORD (51 CITINGS)
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN
Full Text | Citing References

ACCESSION NUMBER: 2005:266919 BIOSIS
DOCUMENT NUMBER: PREV200510053751
TITLE: A **DNA vaccine** targeting **Fos**-related antigen 1 enhanced by IL-18 induces long-lived T-cell memory against tumor recurrence.
AUTHOR(S): Luo, Yunping; Zhou, He; Mizutani, Masato; Mizutani, Noriko; Liu, Cheng; Xiang, Rong; Reisfeld, Ralph A. [Reprint Author]
CORPORATE SOURCE: Scripps Res Inst, Dept Immunol, R218,IMM13,10550 N Torrey

Pines Rd, La Jolla, CA 92037 USA
reisfeld@scripps.edu
SOURCE: Cancer Research, (APR 15 2005) Vol. 65, No. 8, pp. 3419-3427.
CODEN: CNREA8. ISSN: 0008-5472.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Jul 2005
Last Updated on STN: 21 Jul 2005
AB A novel vaccination strategy induced specific CW8(+) T cell-mediated immunity that eradicated spontaneous and experimental pulmonary cancer metastases in syngeneic mice and was also effective in a therapeutic setting of established breast cancer metastases. This was achieved by targeting transcription factor **Fos**-related antigen 1(Fra-1), overexpressed by many tumor cells, with an ubiquitinated **DNA vaccine** against Fra-1, coexpressing secretory IL-18. Insight into the immunologic mechanisms involved was provided by adoptive transfer of T lymphocytes from successfully immunized BALB/c mice to syngeneic severe combined immunodeficient (SCID) mice. Specifically, long-lived T memory cells were maintained dormant in nonlymphoid tissues by IL-18 in the absence of tumor antigen. Importantly, a second tumor cell challenge of these SCID mice restored both, robust tumor-specific cytotoxicity and long-lived T-cell memory, capable of eradicating established pulmonary cancer metastases, suggesting that this vaccine could be effective against tumor recurrence.

Full Text | Citing References

ACCESSION NUMBER: 2003:478104 BIOSIS
DOCUMENT NUMBER: PREV200300478104
TITLE: Transcription factor Fos-related antigen 1 is an effective target for a breast cancer vaccine.
AUTHOR(S): Luo, Yunping; Zhou, He; Mizutani, Masato; Mizutani, Noriko; Reisfeld, Ralph A.; Xiang, Rong [Reprint Author]
CORPORATE SOURCE: Department of Immunology, Scripps Research Institute, La Jolla, CA, 92037, USA
rxiang@scripps.edu
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (July 22 2003) Vol. 100, No. 15, pp. 8850-8855. print.
ISSN: 0027-8424 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Oct 2003
Last Updated on STN: 15 Oct 2003
AB Protection against breast cancer was achieved with a **DNA vaccine** against murine transcription factor **Fos**-related antigen 1, which is overexpressed in aggressively proliferating D2F2 murine breast carcinoma. Growth of primary s.c. tumor and dissemination of pulmonary metastases was markedly suppressed by this oral **DNA vaccine**, carried by attenuated Salmonella typhimurium, encoding murine **Fos**-related antigen 1, fused with mutant polyubiquitin, and cotransformed with secretory murine IL-18. The life span of 60% of vaccinated mice was tripled in the absence of detectable tumor growth after lethal tumor cell challenge. Immunological mechanisms involved activation of T, natural killer, and dendritic cells, as indicated by up-regulation of their activation markers and costimulatory molecules. Markedly increased specific target cell lysis was mediated by both MHC class I-restricted CD8+ T cells and natural killer cells isolated from splenocytes of vaccinated mice, including a significant release of proinflammatory cytokines IFN-gamma and IL-2.

Importantly, fluorescence analysis of fibroblast growth factor 2 and tumor cell-induced vessel growth in Matrigel plugs demonstrated marked suppression of angiogenesis only in vaccinated animals. Taken together, this multi-functional DNA vaccine proved effective in protecting against growth and metastases of breast cancer by combining the action of immune effector cells with suppression of tumor angiogenesis.

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L20 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2010:878637 CAPLUS
TITLE: DNA vaccines for the treatment of prostate cancer
AUTHOR(S): Alam, Sheeba; McNeel, Douglas G.
CORPORATE SOURCE: Department of Medicine, University of Wisconsin Carbone Comprehensive Cancer Center, Madison, WI, USA
SOURCE: Expert Review of Vaccines (2010), 9(7), 731-745
CODEN: ERVXAX; ISSN: 1476-0584
PUBLISHER: Expert Reviews Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB Prostate cancer is a significant public health problem, and the most commonly diagnosed cancer in the USA. The long natural history of prostate cancer, the presence of a serum biomarker that can be used to detect very early recurrences, and the previous identification of multiple potential tissue-specific target antigens are all features that make this disease suitable for the development of anti-tumor vaccines. To date, many anti-tumor vaccines have entered clin. testing for patients with prostate cancer, and some have demonstrated clin. benefit. DNA vaccines represent one vaccine approach that has been evaluated in multiple preclin. models and clin. trials. The safety, specificity for the target antigen, ease of manufg. and ease of incorporating other immune-modulating approaches make DNA vaccines particularly relevant for future development. This article focuses on **DNA vaccines** specifically in the context of prostate cancer treatment, focusing on **antigens** targeted in preclin. models, recent clin. **trials** and efforts to improve the potency of these vaccines.
REFERENCE COUNT: 128 THERE ARE 128 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 2 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2010:453256 CAPLUS
DOCUMENT NUMBER: 153:59723
TITLE: DNA vaccines: developing new strategies against cancer
AUTHOR(S): Fioretti, Daniela; Iurescia, Sandra; Fazio, Vito Michele; Rinaldi, Monica
CORPORATE SOURCE: Institute of Neurobiology and Molecular Medicine, Department of Medicine, National Research Council (CNR), Rome, 00133, Italy
SOURCE: Journal of Biomedicine and Biotechnology (2010) No pp. given
CODEN: JBBOAJ; ISSN: 1110-7251
URL: http://downloads.hindawi.com/journals/jbb/2010/174378.pdf
PUBLISHER: Hindawi Publishing Corp.

DOCUMENT TYPE: Journal; General Review; (online computer file)
LANGUAGE: English
AB A review. Due to their rapid and widespread development, DNA vaccines have entered into a variety of human clin. trials for vaccines against various diseases including cancer. Evidence that DNA vaccines are well tolerated and have an excellent safety profile proved to be of advantage as many clin. trials combines the first phase with the second, saving both time and money. It is clear from the results obtained in clin. **trials** that such **DNA vaccines** require much improvement in **antigen** expression and delivery methods to make them sufficiently effective in the clinic. Similarly, it is clear that addnl. strategies are required to activate effective immunity against poorly immunogenic tumor antigens. Engineering vaccine design for manipulating antigen presentation and processing pathways is one of the most important aspects that can be easily handled in the DNA vaccine technol. Several approaches have been investigated including DNA vaccine engineering, co-delivery of immunomodulatory mols., safe routes of administration, prime-boost regimen and strategies to break the immunosuppressive networks mechanisms adopted by malignant cells to prevent immune cell function. Combined or single strategies to enhance the efficacy and immunogenicity of DNA vaccines are applied in completed and ongoing clin. trials, where the safety and tolerability of the DNA platform are substantiated. In this review on DNA vaccines, salient aspects on this topic going from basic research to the clinic are evaluated. Some representative DNA cancer vaccine studies are also discussed.
REFERENCE COUNT: 149 THERE ARE 149 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References

ACCESSION NUMBER: 2009:1190632 CAPLUS
DOCUMENT NUMBER: 152:546148
TITLE: Safety and immunological efficacy of a DNA vaccine encoding prostatic acid phosphatase in patients with stage D0 prostate cancer
AUTHOR(S): McNeel, Douglas G.; Dunphy, Edward J.; Davies, James G.; Frye, Thomas P.; Johnson, Laura E.; Staab, Mary Jane; Horvath, Dorothea L.; Straus, Jane; Alberti, Dona; Marnocha, Rebecca; Liu, Glenn; Eickhoff, Jens C.; Wilding, George
CORPORATE SOURCE: University of Wisconsin Paul P. Carbone Comprehensive Cancer Center, Madison, WI, USA
SOURCE: Journal of Clinical Oncology (2009), 27(25), 4047-4054
CODEN: JCONDN; ISSN: 0732-183X
PUBLISHER: American Society of Clinical Oncology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Purpose: Prostatic acid phosphatase (PAP) is a prostate tumor antigen. We have previously demonstrated that a DNA vaccine encoding PAP can elicit antigen-specific CD8+ T cells in rodents. We report here the results of a phase I/IIa trial conducted with a DNA vaccine encoding human PAP in patients with stage D0 prostate cancer. Patients and Methods: Twenty-two patients were treated in a dose-escalation trial with 100 μg, 500 μg, or 1,500 μg plasmid DNA, coadministered intradermally with 200 μg granulocyte-macrophage colony-stimulating factor as a vaccine adjuvant, six times at 14-day intervals. All patients were obsd. for 1 yr after treatment. Results: No significant adverse events were obsd. Three (14%) of 22 patients developed PAP-specific IFNγ-secreting CD8+

T-cells immediately after the treatment course, as detd. by enzyme-linked immunospot. Nine (41 %) of 22 patients developed PAP-specific CD4+ and/or CD8+ T-cell proliferation. Antibody responses to PAP were not detected. Overall, the prostate-specific antigen (PSA) doubling time was obsd. to increase from a median 6.5 mo pretreatment to 8.5 mo on-treatment (P = .033), and 9.3 mo in the 1-yr post-treatment period (P = .054). Conclusion: The demonstration that a **DNA vaccine** encoding PAP is safe, elicits an **antigen**-specific T-cell response, and may be assocd. with an increased PSA doubling time suggests that a multi-institutional phase II **trial** designed to evaluate clin. efficacy is warranted.

OS.CITING REF COUNT: 19 THERE ARE 19 CAPLUS RECORDS THAT CITE THIS RECORD (19 CITINGS)

REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 4 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2008:1078606 CAPLUS
DOCUMENT NUMBER: 150:420535
TITLE: A DNA vaccine for multiple sclerosis
AUTHOR(S): Garren, Hideki
CORPORATE SOURCE: Department of Neurology and Neurological Sciences, Stanford University, Stanford, CA, USA
SOURCE: Expert Opinion on Biological Therapy (2008), 8(10), 1539-1550
CODEN: EOBTA2; ISSN: 1471-2598
PUBLISHER: Informa Healthcare
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Background: Multiple sclerosis (MS) is a disease in which safety is of paramount importance when developing a potential therapeutic. Antigen-specific treatments provide a method for achieving efficacy while maintaining safety. **DNA vaccines** are one such form of treatment that have been tested in clin. **trials** Objective: To det. if a **DNA vaccine** is a viable method of **antigen**-specific treatment of MS. Results/conclusion: Phase I and II **trials** of BHT-3009, a **DNA vaccine** encoding myelin basic protein, demonstrated that it was safe, well-tolerated, and caused **antigen**-specific immune tolerance. BHT-3009 showed efficacy in reducing brain lesion activity as well as clin. relapses in patients that were immunol. active at baseline. BHT-3009 is a promising therapy in development for MS, and may prove to be one of the first antigen-specific treatments for this disease.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 95 THERE ARE 95 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 5 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2008:1030371 CAPLUS
DOCUMENT NUMBER: 150:175847
TITLE: The "A, B and C" of Her-2 DNA vaccine development
AUTHOR(S): Wei, Wei-Zen; Jacob, Jennifer; Radkevich-Brown, Olga; Whittington, Paula; Kong, Yi-chi M.
CORPORATE SOURCE: Karmanos Cancer Institute and Department of Immunology and Microbiology, Wayne State University, Detroit, MI, 48201, USA
SOURCE: Cancer Immunology Immunotherapy (2008), 57(11),

1711-1717
CODEN: CIIMDN; ISSN: 0340-7004
PUBLISHER: Springer
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. The development of Her-2 **DNA vaccine** has progressed through three phases that can be categorized as phase "A": the pursuit of Her-2 as a tumor-assocd. **"antigen"**, phase "B": tilting the "balance" between tumor immunity and autoimmunity and phase "C": the on-going "clin. **trials"**. In phase "A", a panel of human ErbB-2 or Her-2 plasmids were constructed to encode non-transforming Her-2 derivs. The immunogenicity and anti-tumor activity of Her-2 DNA vaccines were tested in human Her-2 transgenic mice with or without the depletion of regulatory T cells (Tregs). However, Treg depletion or other immune modulating regimens may increase the risk of autoimmunity. In phase "B", the balance between tumor immunity and autoimmunity was assessed by monitoring the development of exptl. autoimmune thyroiditis (EAT). To test the efficacy of Her-2 DNA vaccines in cancer patients, clin. trials have been initiated in phase "C". Significant anti-Her-2 and anti-tumor activity was obsd. when Her-2 transgenic mice were electro-vaccinated after Treg depletion. Susceptibility to EAT was also enhanced by Treg depletion and there was mutual amplification between Her-2 immunity and EAT development. Although Tregs regulate both EAT and Her-2 immunity, their effector mechanisms may differ. It may be possible to amplify tumor immunity with improved strategies that can bypass undue autoimmunity. Crit. information will be revealed in the next decade to expedite the development of cancer vaccines.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 6 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2008:307551 CAPLUS
DOCUMENT NUMBER: 148:583434
TITLE: DNA vaccination for prostate cancer
AUTHOR(S): Roos, Anna-Karin; King, Alan; Pisa, Pavel
CORPORATE SOURCE: Department of Oncology and Pathology, Cancer Center Karolinska, Karolinska Institute, Stockholm, Swed.
SOURCE: Methods in Molecular Biology (Totowa, NJ, United States) (2008), 423(Electroporation Protocols), 463-472
CODEN: MMBIED; ISSN: 1064-3745
PUBLISHER: Humana Press Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. DNA-based cancer vaccines have been used successfully in mice to induce cytotoxic T lymphocytes (CTLs) specific for prostate antigens. Translation of a prostate-specific **antigen** (PSA) **DNA vaccine** into a phase I clin. **trial** demonstrated that PSA-specific immune responses could be induced but at a significantly lower level compared with those in mice. To enhance the efficacy of DNA vaccination against prostate cancer, we have explored and optimized intradermal electroporation as an effective way of delivering a PSA DNA vaccine. The results demonstrated that intradermal DNA vaccination using low amts. of DNA, followed by two sets of elec. pulses of different length and voltage, effectively induced PSA-specific T cells. Here we describe in detail how to perform intradermal DNA electroporation to induce high gene expression in skin

and, more important, how to induce and analyze PSA-specific T cell responses.

OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 7 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:842719 CAPLUS
DOCUMENT NUMBER: 147:446136
TITLE: Pre-clinical evaluation of a CEA DNA prime/protein boost vaccination strategy against colorectal cancer
AUTHOR(S): Hallermalm, K.; Johansson, S.; Brave, A.; Ek, M.; Engstrom, G.; Boberg, A.; Gudmundsdotter, L.; Blomberg, P.; Mellstedt, H.; Stout, R.; Liu, M. A.; Wahren, B.
CORPORATE SOURCE: Department of Microbiology and Tumor and Cell Biology, Karolinska Institutet and Swedish Institute for Infectious Disease Control, Tualatin, OR, USA
SOURCE: Scandinavian Journal of Immunology (2007), 66(1), 43-51
CODEN: SJIMAX; ISSN: 0300-9475
PUBLISHER: Blackwell Publishing Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In prepn. for a clin. **trial** in patients diagnosed with colorectal cancer, a vaccination strategy targeting the carcinoembryonic **antigen** (CEA) was evaluated in mice using a GMP-produced plasmid **DNA vaccine**, CEA66, encoding a truncated form of the tumor-assocd. **antigen**, CEA. The GMP-produced CEA DNA vaccine was also evaluated for toxicity. Repeated intradermal administration of the GMP-produced vaccine using a novel needle-free jet injection device (Biojector) induced robust CD4 and CD8 T-cell responses in mice, and did not result in any vaccine-related toxicity. In a heterologous DNA prime/protein boost setting, cellular immune responses were of higher magnitude in animals primed with CEA66 DNA than in animals receiving repeated doses of recombinant CEA protein. These responses were further enhanced if recombinant murine granulocyte-macrophage colony-stimulating factor was given as an adjuvant prior to vaccination. In contrast to repeated administration of recombinant CEA protein as a single modality vaccine, the heterologous CEA66 DNA prime/rCEA boost vaccination strategy resulted in a qual. broader immune response, and supports clin. testing of this vaccination regimen in humans.

OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2007:770366 CAPLUS
DOCUMENT NUMBER: 147:196818
TITLE: A vaccine to prevent transmission of human malaria: a long way to travel on a dusty and often bumpy road
AUTHOR(S): Kumar, Nirbhay
CORPORATE SOURCE: Johns Hopkins Malaria Research Institute, Molecular Microbiology and Immunology, Bloomberg School of

Public Health, Johns Hopkins University, Baltimore, MD, 21205, USA
SOURCE: Current Science (2007), 92(11), 1535-1544
CODEN: CUSCAM; ISSN: 0011-3891
PUBLISHER: Current Science Association
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The goal for an effective malaria transmission-blocking vaccine (TBV) is to induce immunity against the stages of the parasite that infect mosquitoes so that malaria transmission can be reduced or halted. Malaria transmission is generally spatially confined to an infectious source, thus a TBV used in a community can effectively suppress malaria transmission to others. Antibodies induced by TBVs target antigens on the surface of sexual and mosquito midgut stages of the malaria parasite and antibodies interfere with the development of the parasites in the midgut of the mosquito. Proteins synthesized in the gametocytes (pre-fertilization antigens, in Plasmodium falciparum: Pfs230 and Pfs48/45) and in the zygotes-ookinetes (post-fertilization antigens, in P. falciparum: Pfs25 and Pfs28) represent some of the key target antigens for the development of TBVs. All the four proteins contain multiple cysteine-rich sequences and the epitopes recognized by transmission-blocking antibodies are redn.-sensitive conformational in nature. The inability to express properly folded proteins has frustrated a protein-based TBV development approach and DNA-based vaccine constructs were envisaged to overcome the conformational problem in recombinant proteins. Indeed studies in mice and monkeys have firmly established the value of DNA-based TBV approach. Although immunogenic in larger animals, delivery of DNA-based TBVs needs to be further optimized to elicit a strong and long lasting functional immune response. This **DNA vaccine** platform can also facilitate evaluation of a cocktail of pre- and post-fertilization **antigens** in pre-clin. setting prior to the development of an ideal and effective TBV for clin. **trials** in human volunteers.
OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)
REFERENCE COUNT: 92 THERE ARE 92 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 9 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:603844 CAPLUS
DOCUMENT NUMBER: 144:348557
TITLE: Carriers for the delivery of a vaccine against respiratory syncytial virus
AUTHOR(S): Cranage, Martin; Taylor, Geraldine
CORPORATE SOURCE: Centre for Infection, Division of Cellular and Molecular Medicine, University of London, London, SW17 0RE, UK
SOURCE: Expert Opinion on Biological Therapy (2005), 5(7), 939-952
CODEN: EOBTA2; ISSN: 1471-2598
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Respiratory syncytial virus (RSV) is a major cause of bronchiolitis and pneumonia in young children and the elderly. Despite its clin. importance, there is no licensed vaccine available at present. Vaccine development has been hampered by observations of increased pathol. after RSV infection in infants vaccinated with formalin-inactivated RSV; incomplete immunity following natural infection; and the need to be

effective during the neonatal period when levels of maternal antibody are high. Four categories of RSV vaccine carriers - live-attenuated RSVs, recombinant vectors expressing the protective **antigens** of RSV, **DNA vaccines** and subunit vaccines - have been evaluated in animal models and/or clin. **trials**. So far, studies with live-attenuated virus vaccines highlight the need to improve immunogenicity while maintaining a suitable level of attenuation. Studies with recombinant vectors, DNA and subunit vaccines illustrate the pivotal nature of the vaccine carrier in detg. the balance between immune-mediated protection against infection and the induction of immune-mediated pulmonary pathol.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)

REFERENCE COUNT: 148 THERE ARE 148 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 10 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:385528 CAPLUS
DOCUMENT NUMBER: 142:409267
TITLE: Tumor antigens for cancer immunotherapy: Therapeutic potential of xenogeneic DNA vaccines
AUTHOR(S): Srinivasan, Roopa; Wolchok, Jedd D.
CORPORATE SOURCE: Division of Tumor Immunology, Dept. of Research, CancerVax Corporation, Carlsbad, CA, 92008, USA
SOURCE: Journal of Translational Medicine (2004), 2, No pp. given
CODEN: JTMOBV; ISSN: 1479-5876
URL: http://www.translational-medicine.com/content/pdf/1479-5876-2-12.pdf
PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; General Review; (online computer file)
LANGUAGE: English

AB A review. Preclin. animal studies have convincingly demonstrated that tumor immunity to self antigens can be actively induced and can translate into an effective anti-tumor response. Several of these observations are being tested in clin. trials. Immunization with xenogeneic DNA is an attractive approach to treat cancer since it generates T cell and antibody responses. When working in concert, these mechanisms may improve the efficacy of vaccines. The use of xenogeneic DNA in overcoming immune tolerance has been promising not only in inbred mice with transplanted tumors but also in outbred canines, which present with spontaneous tumors, as in the case of human. Use of this strategy also overcomes limitations seen in other types of cancer vaccines. Immunization against defined tumor **antigens** using a xenogeneic **DNA vaccine** is currently being tested in early phase clin. **trials** for the treatment of melanoma and prostate cancers, with proposed **trials** for breast cancer and Non-Hodgkin's Lymphoma.

REFERENCE COUNT: 97 THERE ARE 97 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN

Full Text | Citing References

ACCESSION NUMBER: 2005:113893 CAPLUS
DOCUMENT NUMBER: 142:196372
TITLE: Enhanced immunogenicity using an alphavirus replicon DNA vaccine against human immunodeficiency virus type 1

AUTHOR(S): Nordstroem, Eva K. L.; Forsell, Mattias N. E.; Barnfield, Christina; Bonin, Eivor; Hanke, Tomas; Sundstroem, Magnus; Karlsson, Gunilla B.; Liljestroem, Peter
CORPORATE SOURCE: Microbiology and Tumor Biology Center, Karolinska Institute, Stockholm, S-171 77, Swed.
SOURCE: Journal of General Virology (2005), 86(2), 349-354 CODEN: JGVIAY; ISSN: 0022-1317
PUBLISHER: Society for General Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB With the human immunodeficiency virus type 1 (HIV-1) epidemic expanding at increasing speed, development of a safe and effective vaccine remains a high priority. One of the most central vaccine platforms considered is plasmid DNA. However, high doses of DNA and several immunizations are typically needed to achieve detectable T-cell responses. In this study, a Semliki Forest virus replicon **DNA vaccine** designed for human clin. **trials**, DREP.HIVA, encoding an **antigen** that is currently being used in human **trials** in the context of a conventional DNA plasmid, pTHr.HIVA, was generated. It was shown that a single immunization of DREP.HIVA stimulated HIV-1-specific T-cell responses in mice, suggesting that the poor immunogenicity of conventional DNA vaccines may be enhanced by using viral replicon-based plasmid systems. The results presented here support the evaluation of Semliki Forest virus replicon DNA vaccines in non-human primates and in clin. studies.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 12 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2000:672733 CAPLUS
DOCUMENT NUMBER: 134:264623
TITLE: DNA vaccines against B-cell tumors
AUTHOR(S): Stevenson, Freda K.; Zhu, Delin; Spellerberg, Myfanwy; King, Catherine A.; Sahota, Surinder S.; Rice, Jason; Thompsett, Andrew R.; Hamblin, Terry J.
CORPORATE SOURCE: Molecular Immunology Group Tenovus Laboratory, Southampton University Hospital Trust, Southampton, UK
SOURCE: Cancer Vaccines and Immunotherapy (2000), 218-236. Editor(s): Stern, Peter L.; Beverley, Peter C. L.; Carroll, Miles W. Cambridge University Press: Cambridge, UK. CODEN: 69AKYN
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review with 55 refs. Topics discussed include B-cell malignancies and the clin. need for vaccines, idiotypic determinants as B-cell tumor **antigens**, **DNA vaccines**, and clin. **trials** of **DNA vaccines**.
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 13 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 2000:549736 CAPLUS
DOCUMENT NUMBER: 134:176875
TITLE: DNA vaccination against cancer antigens
AUTHOR(S): Stevenson, F. K.; Zhu, D.; Spellerberg, M. B.; Rice, J.; King, C. A.; Thompsett, A. R.; Sahota, S. S.;

Hamblin, T. J.
CORPORATE SOURCE: Molecular Immunology Group, Tenovus Laboratory, Southampton University Hospital, Southampton, SO9 4XY, UK
SOURCE: Ernst Schering Research Foundation Workshop (2000), 30(Therapeutic Vaccination Strategies), 119-136
CODEN: ESRWEL; ISSN: 0947-6075
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 32 refs. Topics discussed include a general overview of **DNA vaccines**, idiotypic Ig of B cell malignancies, idiotypic **DNA vaccines** against lymphoma, DNA fusion gene vaccines against alternative tumor **antigens**, idiotypic DNA fusion gene vaccines against secreted Ig of multiple myeloma, effect of pre-existing anti-fragment C (anti-FrC) antibody, clin. **trial** of **DNA vaccines**, and **DNA vaccines** against intracellular tumor **antigens**.
OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 14 OF 23 CAPLUS COPYRIGHT 2011 ACS on STN
Full Text | Citing References
ACCESSION NUMBER: 1997:726095 CAPLUS
DOCUMENT NUMBER: 128:2681
ORIGINAL REFERENCE NO.: 128:595a,598a
TITLE: Toward clinical trials of DNA vaccines against malaria
AUTHOR(S): Hoffman, Stephen L.; Doolan, Denise L.; Sedegah, Martha; Wang, Ruobing; Scheller, Libia F.; Kumar, Anita; Weiss, Walter R.; Le, Thong P.; Klinman, Dennis M.; Hobart, Peter; Norman, Jon A.; Hedstrom, Richard C.
CORPORATE SOURCE: Malaria Program, Naval Med. Res. Inst., Bethesda, MD, USA
SOURCE: Immunology and Cell Biology (1997), 75(4), 376-381
CODEN: ICBIEZ; ISSN: 0818-9641
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 65 refs. In mid 1997 the first malaria DNA vaccine has entered clin. trials. This single gene DNA vaccine encoding the Plasmodium falciparum circumsporozoite protein (PfCSP) will be studied for safety and immunogenicity. If these criteria are met, a multi-gene DNA vaccine designed to induce protective CD8+ T cell responses against P. falciparum infected hepatocytes will be subsequently assessed for safety, immunogenicity, and capacity to protect immunized volunteers against exptl. challenge with P. falciparum sporozoites. Herein, the authors discuss the rationale and exptl. foundation for these anticipated P. falciparum DNA vaccine trials.
OS.CITING REF COUNT: 22 THERE ARE 22 CAPLUS RECORDS THAT CITE THIS RECORD (22 CITINGS)
REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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STN

ACCESSION NUMBER: 2010:328250 BIOSIS
DOCUMENT NUMBER: PREV201000328250
TITLE: DNA Vaccines: Developing New Strategies against Cancer.
AUTHOR(S): Fioretti, Daniela; Iurescia, Sandra; Fazio, Vito Michele; Rinaldi, Monica [Reprint Author]
CORPORATE SOURCE: CNR, Dept Med, Inst Neurobiol and Mol Med, Via Fosso Cavaliere 100, I-00133 Rome, Italy
monica.rinaldi@artov.inmm.cnr.it
SOURCE: Journal of Biomedicine & Biotechnology, (2010) pp. Article No.: 174378.
ISSN: 1110-7243.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Jun 2010
Last Updated on STN: 9 Jun 2010

AB Due to their rapid and widespread development, DNA vaccines have entered into a variety of human clinical trials for vaccines against various diseases including cancer. Evidence that DNA vaccines are well tolerated and have an excellent safety profile proved to be of advantage as many clinical trials combines the first phase with the second, saving both time and money. It is clear from the results obtained in clinical **trials** that such **DNA vaccines** require much improvement in **antigen** expression and delivery methods to make them sufficiently effective in the clinic. Similarly, it is clear that additional strategies are required to activate effective immunity against poorly immunogenic tumor antigens. Engineering vaccine design for manipulating antigen presentation and processing pathways is one of the most important aspects that can be easily handled in the DNA vaccine technology. Several approaches have been investigated including DNA vaccine engineering, co-delivery of immunomodulatory molecules, safe routes of administration, prime-boost regimen and strategies to break the immunosuppressive networks mechanisms adopted by malignant cells to prevent immune cell function. Combined or single strategies to enhance the efficacy and immunogenicity of DNA vaccines are applied in completed and ongoing clinical trials, where the safety and tolerability of the DNA platform are substantiated. In this review on DNA vaccines, salient aspects on this topic going from basic research to the clinic are evaluated. Some representative DNA cancer vaccine studies are also discussed.

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ACCESSION NUMBER: 2008:387184 BIOSIS
DOCUMENT NUMBER: PREV200800387183
TITLE: DNA vaccination for prostate cancer.
AUTHOR(S): Roos, Anna-Karin [Reprint Author]; King, Alan; Pisa, Pavel
CORPORATE SOURCE: Karolinska Inst, Canc Ctr Karolinska R8 01, Dept Pathol and Oncol, Stockholm, Sweden
SOURCE: Li, S [Editor]. Methods in Molecular Biology, (2008) pp. 463-472. Methods in Molecular Biology.
Publisher: HUMANA PRESS INC, 999 RIVERVIEW DR, STE 208, TOTOWA, NJ 07512-1165 USA. Series: METHODS IN MOLECULAR BIOLOGY.
ISSN: 1064-3745. ISBN: 978-1-58829-877-5(H).
DOCUMENT TYPE: Book; (Book Chapter)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jul 2008
Last Updated on STN: 16 Jul 2008

AB DNA-based cancer vaccines have been used successfully in mice to induce cytotoxic T lymphocytes (CTLs) specific for prostate antigens. Translation of a prostate-specific **antigen** (PSA) **DNA vaccine** into a phase I clinical **trial** demonstrated that PSA-specific immune responses could be induced but at a significantly lower level compared with those in mice. To enhance the efficacy of DNA vaccination against prostate cancer, we have explored and optimized intradermal electroporation as an effective way of delivering a PSA DNA vaccine. The results demonstrated that intradermal DNA vaccination using low amounts of DNA, followed by two sets of electrical pulses of different length and voltage, effectively induced PSA-specific T cells. Here we describe in detail how to perform intradermal DNA electroporation to induce high gene expression in skin and, more important, how to induce and analyze PSA-specific T cell responses.

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STN

ACCESSION NUMBER: 2008:199957 BIOSIS
DOCUMENT NUMBER: PREV200800193803
TITLE: **DNA vaccine trials** using 3 blood stage **antigens** of Plasmodium vivax Korean isolates.
AUTHOR(S): Shin, Eun-Hee [Reprint Author]; Kim, Hyo-Jin; Lee, Jo Woon Yi; Lee, Jin-Ju; Chai, Jong-Yil
CORPORATE SOURCE: Seoul Natl Univ, Coll Med, Seoul 151, South Korea
SOURCE: American Journal of Tropical Medicine and Hygiene, (NOV 2007) Vol. 77, No. 5, Suppl. S, pp. 163.
Meeting Info.: 56th Annual Meeting of the American-Society-of-Tropical-Medicine-and-Hygiene. Philadelphia, PA, USA. November 04 -08, 2007. Amer Soc Trop Med & Hyg.
CODEN: AJTHAB. ISSN: 0002-9637.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Mar 2008
Last Updated on STN: 19 Mar 2008

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STN

ACCESSION NUMBER: 2008:5477 BIOSIS
DOCUMENT NUMBER: PREV200800004390
TITLE: A vaccine to prevent transmission of human malaria: A long way to travel on a dusty and often bumpy road.
AUTHOR(S): Kumar, Nirbhay [Reprint Author]
CORPORATE SOURCE: Johns Hopkins Med Inst, Bloomberg Sch Publ Hlth, Johns Hopkins Malaria Res Ctr, 615 N Wolfe St, Baltimore, MD 21205 USA
nkumar@jhsph.edu
SOURCE: Current Science (Bangalore), (JUN 10 2007) Vol. 92, No. 11, pp. 1535-1544.
CODEN: CUSCAM. ISSN: 0011-3891.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 2007
Last Updated on STN: 12 Dec 2007
AB The goal for an effective malaria transmission-blocking vaccine (TBV) is

to induce immunity against the stages of the parasite that infect mosquitoes so that malaria transmission can be reduced or halted. Malaria transmission is generally spatially confined to an infectious source, thus a TBV used in a community can effectively suppress malaria transmission to others. Antibodies induced by TBVs target antigens on the surface of sexual and mosquito midgut stages of the malaria parasite and antibodies interfere with the development of the parasites in the midgut of the mosquito. Proteins synthesized in the gametocytes (pre-fertilization antigens, in Plasmodium falciparum: Pfs230 and Pfs48/45) and in the zygotes-ookinetes (post-fertilization antigens, in P. falciparum: Pfs25 and Pfs28) represent some of the key target antigens for the development of TBVs. All the four proteins contain multiple cysteine-rich sequences and the epitopes recognized by transmission-blocking antibodies are reduction-sensitive conformational in nature. The inability to express properly folded proteins has frustrated a protein-based TBV development approach and DNA-based vaccine constructs were envisaged to overcome the conformational problem in recombinant proteins. Indeed studies in mice and monkeys have firmly established the value of DNA-based TBV approach. Although inummogenic in larger animals, delivery of DNA-based TBVs needs to be further optimized to elicit a strong and long lasting functional immune response. This **DNA vaccine** platform can also facilitate evaluation of a cocktail of pre- and post-fertilization **antigens** in pre-clinical setting prior to the development of an ideal and effective TBV for clinical **trials** in human volunteers.

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STN

ACCESSION NUMBER: 2007:466754 BIOSIS
DOCUMENT NUMBER: PREV200700472517
TITLE: Pre-clinical evaluation of a CEA DNA prime/protein boost vaccination strategy against colorectal cancer.
AUTHOR(S): Hallermalm, K. [Reprint Author]; Johansson, S.; Brave, A.; Ek, M.; Engstrom, G.; Boberg, A.; Gudmundsdotter, L.; Blomberg, P.; Mellstedt, H.; Stout, R.; Liu, M. A.; Wahren, B.
CORPORATE SOURCE: Swedish Inst Infect Dis Control, Dept Virol Immunol and Vaccinol, SE-17182 Solna, Sweden
kristian.hallermalm@smi.ki.se
SOURCE: Scandinavian Journal of Immunology, (JUL 2007) Vol. 66, No. 1, pp. 43-51.
CODEN: SJIMAX. ISSN: 0300-9475.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Sep 2007
Last Updated on STN: 5 Sep 2007

AB In preparation for a clinical **trial** in patients diagnosed with colorectal cancer, a vaccination strategy targeting the carcinoembryonic **antigen** (CEA) was evaluated in mice using a GMP-produced plasmid **DNA vaccine**, CEA66, encoding a truncated form of the tumour-associated **antigen**, CEA. The GMP-produced CEA DNA vaccine was also evaluated for toxicity. Repeated intradermal administration of the GMP-produced vaccine using a novel needle-free jet injection device (Biojector) induced robust CD4 and CD8 T-cell responses in mice, and did not result in any vaccine-related toxicity. In a heterologous DNA prime/protein boost setting, cellular immune responses were of higher magnitude in animals primed with CEA66 DNA than in animals receiving repeated doses of recombinant CEA protein. These responses were further enhanced if recombinant murine granulocyte-macrophage colony-stimulating factor was

given as an adjuvant prior to vaccination. In contrast to repeated administration of recombinant CEA protein as a single modality vaccine, the heterologous CEA66 DNA prime/rCEA boost vaccination strategy resulted in a qualitatively broader immune response, and supports clinical testing of this vaccination regimen in humans.

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STN

ACCESSION NUMBER: 2005:479124 BIOSIS
DOCUMENT NUMBER: PREV200510271028
TITLE: DNA immunization against melanoma antigens enhances tumor immunity in mice following sub-lethal irradiation and immune reconstitution.
AUTHOR(S): Diab, Adi [Reprint Author]; Hubbard, Vanessa M.; Cohen, Adam; Huggins, Deonka; Kochman, Adam; Guevarra, Jose A.; Engelhorn, Manuel; Wolchok, Jedd D.; van den Brink, Marcel; Houghton, Alan N.; Perales, Miguel-Angel
CORPORATE SOURCE: Mem Sloan Kettering Canc Ctr, Dept Med, New York, NY 10021 USA
SOURCE: Blood, (NOV 16 2004) Vol. 104, No. 11, Part 1, pp. 835A. Meeting Info.: 46th Annual Meeting of the American-Society-of-Hematology. San Diego, CA, USA. December 04 -07, 2004. Amer Soc Hematol. CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

AB The development of successful cancer vaccines is contingent on the ability to induce effective anti-tumor immunity against self-antigens that do not typically elicit immunity. We are working on strategies to overcome immunologic tolerance/ignorance to cancer through the use of gene products closely related to self-**antigens**, including xenogeneic DNA and mutated DNA, and have initiated clinical **trials** of **DNA vaccines** in patients with advanced melanoma or prostate cancer. Recent studies have demonstrated that homeostasis-driven T-cell proliferation in the reconstituted lymphodepleted host could enhance the efficacy of whole-cell tumor vaccines. We hypothesized that immunization of sub-lethally irradiated and immune reconstituted mice against specific tumor antigens could increase anti-tumor immunity. B6 mice were irradiated with 600 cGy and immediately reconstituted with 30 x 10(6) syngeneic splenocytes. Weekly DNA immunization using either human tyrosinase-related protein (TRP)-2 DNA (xenogeneic melanoma differentiation antigen (MDA) or Opt-Tyrp1 DNA (a mutated MDA, which has been optimized for CD8 epitopes), was begun on day 1. We have found that: (1) by day 14 after irradiation and reconstitution, recipients have considerable numbers of splenic T cells, including de novo generated donor T cells, which suggests that vaccination aimed at T cells might be feasible; (2) DNA immunization against a single tumor antigen can provide protection from a tumor challenge that is significantly greater than that observed in immunized non-irradiated hosts (Figure); (3) DNA immunization induces higher levels of tumor-specific CD8(+) T cells in the irradiated and reconstituted recipients(detected by intracellular cytokine flow cytometry assay); and (4) the effects of DNA immunization after lymphodepletion with immune reconstitution on bothtumor-free survival and CD8(+) T cell responses have been validated for two different DNA vaccine strategies (TRP-2 DNA and Opt-Tyrp1 DNA). These results demonstrate that DNA immunization following

sub-lethal irradiation andimmune reconstitution can induce potent anti-tumor effects. Furthermore, theyprovide a strong rationale for the development of novel therapeutic strategies that combine lymphodepletion with immune reconstitution and DNA immunizationin human clinical trials.[GRAPHICS]

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STN

ACCESSION NUMBER: 2005:476372 BIOSIS
DOCUMENT NUMBER: PREV200510268276
TITLE: DNA immunization against melanoma antigens enhances tumor immunity in mouse models of allogeneic hematopoietic stem cell transplantation (HSCT).
AUTHOR(S): Diab, Adi [Reprint Author]; Perales, Miguel-Angel; Cohen, Adam; Hubbard, Vanessa M.; Eng, Jeff; Huggins, Deonka; Engelhorn, Manuel; Guevarra, Jose A.; Alpdogan, Onder; Wolchok, Jedd D.; Kochman, Adam; Houghton, Alan N.; van den Brink, Marcel
CORPORATE SOURCE: Mem Sloan Kettering Canc Ctr, Dept Med, New York, NY 10021 USA
SOURCE: Blood, (NOV 16 2004) Vol. 104, No. 11, Part 1, pp. 90A-91A. Meeting Info.: 46th Annual Meeting of the American-Society-of-Hematology. San Diego, CA, USA. December 04 -07, 2004. Amer Soc Hematol. CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Nov 2005
Last Updated on STN: 16 Nov 2005

AB Allogeneic HSCT is an important therapy with curative potential for a variety of malignant diseases, including leukemias, lymphomas and some solid tumors. Despite significant progress in reducing treatment-related mortality, malignantrelapse remains a major problem. We are developing **DNA vaccines** that encode gene products closely related to self-**antigens**, including xenogeneic DNA and mutated DNA, and have initiated clinical **trials** of **DNA vaccines** in patients withadvanced melanoma or prostate cancer. Using the B 16 mouse melanoma model, wehave shown that immunization with human TRP-2 DNA (xenogeneic melanoma differentiation antigen-MDA) or Opt-Tytp1 DNA (a mutated MDA related to TRP-2, whichwe have optimized for CD8 epitopes), can induce tumor protection, including against established tumors. We hypothesized that immunization of allogeneic HSCT recipients (or their donors) against specific tumor antigens could decrease the risk of relapse without enhancing graft-versus-host disease (GVHD). In an MHC-matched minor antigen-mismatched mouse HSCT model (LP into B6), we found that: (1) by day 28 after transplant, recipients of an allogeneic T-cell depleted (TCD)-HSCT have considerable numbers of splenic T cells, including de novo generated donor T cells, which suggests that vaccination aimed at T cells might be feasible; (2) post-HSCT DNA immunization against a single tumor antigen can provide protection from a tumor challenge that is comparable to that observed with a whole cell vaccine (B16-GM-CSF) and significantly greater than HSCT alone; (3) DNA immunization post-HSCT can induce tumor-specific CD8(+) T cells of donor origin (detected by ELISPOT or intracellular cytokine flow cytometry assay); (4) the combination of donor leukocyte infusion (DLI) and post-HSCT DNA immunization further enhances tumor-free survival (Figure); (5) there is no evidence of GVHD in multiple experiments using a clinical GVHD scoreto monitor recipients; and (6) the

effects of post-HSCT DNA immunization on both tumor-free survival and CD8(+) T cell responses have been validated for two different DNA vaccine strategies (hTRP-2 + GM-CSF DNA, or Opt-Tyrp1DNA). These results demonstrate that DNA immunization after allogeneic TCD-HSCT can induce potent anti-tumor effects without the induction of GVHD. This and similar investigations provide a strong rationale for the development of novel therapeutic strategies that combine allogeneic HSCT, post-transplant tumor vaccination and adoptive cell therapy in human clinical trials.[GRAPHICS]

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STN

ACCESSION NUMBER: 2005:169595 BIOSIS
DOCUMENT NUMBER: PREV200500170549
TITLE: Enhanced immunogenicity using an alphavirus replicon DNA vaccine against human immunodeficiency virus type 1.
AUTHOR(S): Nordstrmom, Eva K. L.; Forsell, Mattias N. E.; Barnfield, Christina; Bonin, Eivor; Hanke, Tomas; Sundstrom, Magnus; Karlsson, Gunilla B. [Reprint Author]; Liljestrom, Peter
CORPORATE SOURCE: Ctr Microbiol and Tumor Biol, Karolinska Inst, S-17177, Stockholm, Sweden
Nilla.Karlsson@mtc.ki.se
SOURCE: Journal of General Virology, (February 2005) Vol. 86, No. Part 2, pp. 349-354. print.
ISSN: 0022-1317 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 May 2005
Last Updated on STN: 4 May 2005

AB With the human immunodeficiency virus type I (HIV-1) epidemic expanding at increasing speed, development of a safe and effective vaccine remains a high priority. One of the most central vaccine platforms considered is plasmid DNA. However, high doses of DNA and several immunizations are typically needed to achieve detectable T-Gell responses. In this study, a Semliki Forest virus replicon **DNA vaccine** designed for human clinical **trials**, DREP.HIVA, encoding an **antigen** that is currently being used in human **trials** in the context of a conventional DNA plasmid, pTHr.HIVA, was generated. It was shown that a single immunization of DREP.HIVA stimulated HIV-1-specific T-cell responses in mice, suggesting that the poor immunogenicity of conventional DNA vaccines may be enhanced by using viral replicon-based plasmid systems. The results presented here support the evaluation of Semliki Forest virus replicon DNA vaccines in non-human primates and in clinical studies.

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STN

ACCESSION NUMBER: 2003:561994 BIOSIS
DOCUMENT NUMBER: PREV200300562038
TITLE: DNA vaccines: An active immunization strategy for prostate cancer.
AUTHOR(S): Wolchok, Jedd D. [Reprint Author]; Gregor, Polly D.; Nordquist, Luke T.; Slovin, Susan F.; Scher, Howard I.
CORPORATE SOURCE: Clinical Immunology and Genitourinary Oncology Services, Memorial Sloan-Kettering Cancer Center, 1275 York Ave, New York, NY, 10021, USA
SOURCE: Seminars in Oncology, (October 2003) Vol. 30, No. 5, pp. 659-666. print.

ISSN: 0093-7754 (ISSN print).
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 26 Nov 2003
Last Updated on STN: 26 Nov 2003

=> **transcriptional (w) factor**
L21 8161 TRANSCRIPTIONAL (W) FACTOR

=> **(co-expression) (s) antigen**
L22 631 (CO-EXPRESSION) (S) ANTIGEN

=> **L21 and L22**
L23 1 L21 AND L22

=> **D L23 IBIB ABS**

L23 ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN

Full Text | Citing References

ACCESSION NUMBER: 2006:183871 BIOSIS
DOCUMENT NUMBER: PREV200600185983
TITLE: Differential patterns of transcriptional protein expression may explain functional differences between hematopoietic progenitors derived from human ESC's and fetal hematopoietic tissues.
AUTHOR(S): Shaaban, Aimen F. [Reprint Author]; Golos, Thaddeus G.; Thomson, James A.; Rajesh, Deepika
CORPORATE SOURCE: Univ Wisconsin, Sch Med, Dept Surg Anat and Obstet and Gynecol, Madison, WI USA
SOURCE: Blood, (NOV 16 2005) Vol. 106, No. 11, Part 1, pp. 1007A. Meeting Info.: 47th Annual Meeting of the American-Society-of-Hematology. Atlanta, GA, USA. December 10 -13, 2005. Amer Soc Hematol.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Mar 2006
Last Updated on STN: 15 Mar 2006

AB Recent studies suggest that development of human embryoid body-derived hematopoietic progenitor cells (EB-HPC's) mirrors that of primitive yolk sac-derived hematopoietic progenitors (YS-HPC's) in their expression of transcription factors known to be critically associated with hematopoietic development. However, the findings of these studies were limited to molecular characterization of heterogeneous EB cultures rather than analysis of hematopoietic lineage-committed cell subsets. We wondered: 1) if the expression patterns were consistent at the protein level in both bulk EB cultures and subsets of hematopoietic-lineage committed cells: and 2) if these expression patterns differed from those found in definitive HPC's harvested from early fetal hematopoietic tissues. To answer these questions, we compared the intracellular expression of critical transcriptional regulatory proteins from human and rhesus EB-derived HPC's (hEB-HPC's and rEB-HPC's) with hematopoietic progenitors from rhesus fetal liver (rFL-HPC's) and rhesus placental blood HPC's (rPB-HPC's). Rhesus macaque fetal tissue was used in lieu of scarce human fetal tissues. Using multi-color intracellular flow cytometry we correlated the expression of transcriptional proteins (SCL/TAL1, GATA-1, GATA-2, Oct-3/4,

HOXB4) with cell surface determinants of hematopoietic commitment (CD34, CD31, CD45) in hEB-HPC's, rEB-HPC's, rFL-HPC's and rPB-HPC's. Human and rhesus EB16 cells were harvested from identical culture conditions and compared to 0.34 gestation rFL-HPC's and rPB-HPC's. The frequency of CD45+ cells was higher in hEB-HPC's (31%) and rPB-HPC's (46%) compared to rFL-HPC's and rEB-HPC's. A much higher frequency of GATA-1 and HOXB4 expressing cells was seen in both of the fetal cell types when compared to either rhesus or human EB's. Additionally, the frequency of GATA-2 and SCL expressing cells was comparable in all cell types. Lastly, the expression of Oct3/4 was higher in rPB-HPC's and rFL-HPC's when compared to bulk EB cultures. However, analysis of subsets of EB cells expressing hematopoietic **antigens** (CD45, CD34) revealed **co-expression** of Oct-3/4 on both CD45+ and CD34+ cell fractions. From these findings, we conclude: 1) critical differences exist in GATA-1 and HOXB4 expression between HPC's derived from EB cultures and HPC's harvested during early definitive fetal hematopoietic development; and 2) human EB-derived hematopoietic progenitor cells express persistently high levels of Oct-3/4. The differences in the expression of these critical **transcriptional factors** at the protein level may explain the observed functional differences between EB-derived HPC's and definitive HPC's from fetal and adult sources.

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